CHRONIC SUBDURAL HEMATOMA OF THE CEREBRUM

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IT WAS before the Society of Neurological Surgeons in 1925 that Cushing[1] said,

Certain members of this Society in their desire to advance information concerning a poorly understood and supposedly rare intracranial lesion have generously pooled their experiences in regard to it, so that a single individual might have at his command sufficient material for a comprehensive study. Whether such a novel and unselfish program will lead to anything beyond the present report can not be foreseen, but it at least sets a good precedent.

This pooling of cases referred to by Cushing consisted of five cases from his clinic, two cases from Adson's service, two cases from Naffziger's and one from Sachs' clinic. These ten cases were used by Putnam[1] in his comparative study of chronic subdural hematomas and the cases of pachymeningitis hemorrhagica interna. The histology of these latter cases was quite well established and clarified by Virchow in 1857. The "novel and unselfish program" by the Society of Neurological Surgeons terminated with Putnam's thorough coverage of the subject up to 1925 and aside from an exchange of personal ideas and experiences, the problem of chronic subdural hematoma has not since been officially discussed before that organization.

It is not to be implied that selfishness is the explanation for the failure of subsequent pooling of individuals' cases, but rather, that soon after 1925 various neurosurgeons had a sufficient number of cases of their own to permit conclusions and contributions upon the subject. These contributions began with Rand's[2] study of seven surgical cases with one fatality, followed by Grant's[3] report of three patients who completely recovered following operation.

Jelsma[4] in 1930 reported two cases and analyzed the symptoms of forty-two cases from the literature. There was a death rate of 19.3 per cent following operation in the forty-four cases covered by Jelsma. Fleming and Jones[5] in 1932 suggested double trephine drainages of the subdural hematomas and reported one death in eight cases so treated. Following Fleming and Jones' report, McKenzie[6] reported eleven cases. Five of these were operated upon through osteoplastic flaps and six with small bone openings. He had one fatality. Keegan[7] in 1933 had five cases with one death, citing the stormy convalescences he struggled against. In 1935 Coleman[8] reported twenty-four cases with four deaths. He stressed again the importance of bilateral trephine exploration and a small bony opening for drainage. Gardner[9] followed Coleman with a study of twenty cases in 1935, and added fifteen more cases in 1939. One case of Gardner's was an autopsy study, twenty cases were treated with craniotomies and with no fatalities. He reported two deaths in fourteen of his cases treated with trephine drainages. Furlow[10] in 1936, from Sach's clinic, reported sixteen cases with ten recoveries and concluded that trephine drainage was most applicable in the comatose patient. More recently Ingram[11] has contributed fourteen craniotomies carried out in eleven cases of subdural hematomas in children with only one fatality. Most recently Kunkel and Dandy[12] made a study of forty-eight cases operated upon by Dandy. In this series there were two deaths, sixteen patients had postoperative hemorrhages and five had to be reopened more than one time.

It would be short of sacrilegious to omit Trotter's[13] contribution in 1914 on the subject of subdural hematomas. He reported four cases that he operated upon. He stressed the role of trauma as the etiologic factor, recognized the true histologic changes, and advocated small bone exposures for drainage in lieu of an extensive osteoplastic operation. Aside from anatomic physiologic explanations of the pupillary changes, the ipsilateral motor symptoms, and experimental investigations as to the character and development of the subdural membranes, essentially nothing new has been added to the surgery of subdural hematomas since Trotter's paper. The contributions of Munro[14] and the volume of work done by him on subdural hematomas should not go without due recognition. In that he adheres to the idea of abandoning "the artificial segregation of certain of the so-called chronic forms of this disease," this does not allow for the adherence to a somewhat routine surgical approach as had been applicable in twenty-three of the twenty-four cases in this report. In all of the twenty-four cases reported here there has been an organized subdural, as well as an organized extra-
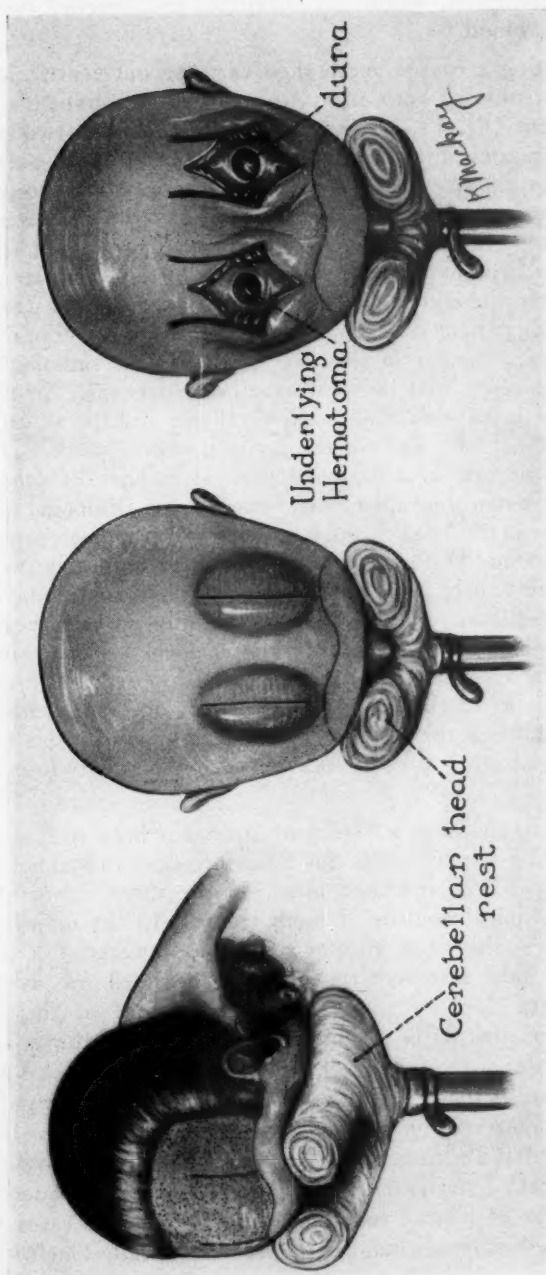
Fig. 1. Only in two of the twenty-three cases trephined for ventriculography has it been necessary to inject air for localization of the hematoma.

arachnoidal membrane which has enveloped the liquefied by-prod-
ucts of the old blood.

Since adopting a routine method of carrying out ventricular air
injections[15], trephining both frontal bones with the patient's fore-
head dependent (fig. 1), it has only been necessary in two of the
twenty-four cases to inject air for the localization of the subdural
hematoma. In the one case in which an osteoplastic exposure of
the hematoma was carried out, it was obvious from the extensive-
ness of the hematoma that had trephine openings in the frontal
bones been made preparatory for ventricular air injection, drainage
of the hematoma could have been effected through the trephine
opening and the patient saved the ordeal of a prolonged craniotomy.
The ventricular changes in the two cases studied radiologically,
with air in the ventricular cavities, have been identical (fig. 1). In
both cases the hematoma was a unilateral one and the ventricular
defects were those of a shift of the cavities, with a maintenance of
the normal contours, in a flattened vertical manner. In the two
cases where ventriculography was resorted to, drainage of the
hematoma was carried out through an enlarged trephine opening in
the temporal bone. With the exception of the craniotomy case the
remaining twenty-three have been drained through enlargements of
the frontal trephines. The underlying condition in twenty-one of
the cases was exposed when the dura was opened preparatory to
introducing the ventricular needles for the proposed air injection.
Further details as to the surgical procedures, once the hematoma
was viewed through the small dural openings, will be dealt with
after relating some of the statistical facts of the twenty-four cases
in this series.

In nineteen of the cases a history of injury has been elicited from
the patients. In a few instances this history has been obtained after
the patients had been operated upon. In the three infants there
were definite clinical records of birth trauma. In the majority of
the adult cases, the head injuries experienced were of a trivial
character. In most instances the blow to the head was received
in a fall to the floor or the ground and in none of these was
there any frank loss of consciousness at the time of the fall. Six
patients gave a history of an automobile accident, only one of which
was thrown out of the car, and again, not one of these six patients
suffered from more than a transitory loss of consciousness. In one
patient who suffered with purpura hemorrhagica, the only traumatic
history was that of strangulation to a degree of facial cyanosis but
not to a degree of loss of consciousness. In only two cases there
was no definite history elicited of trauma to the head. One of these
patients was an artificial basophilism (clinically) and the slightest
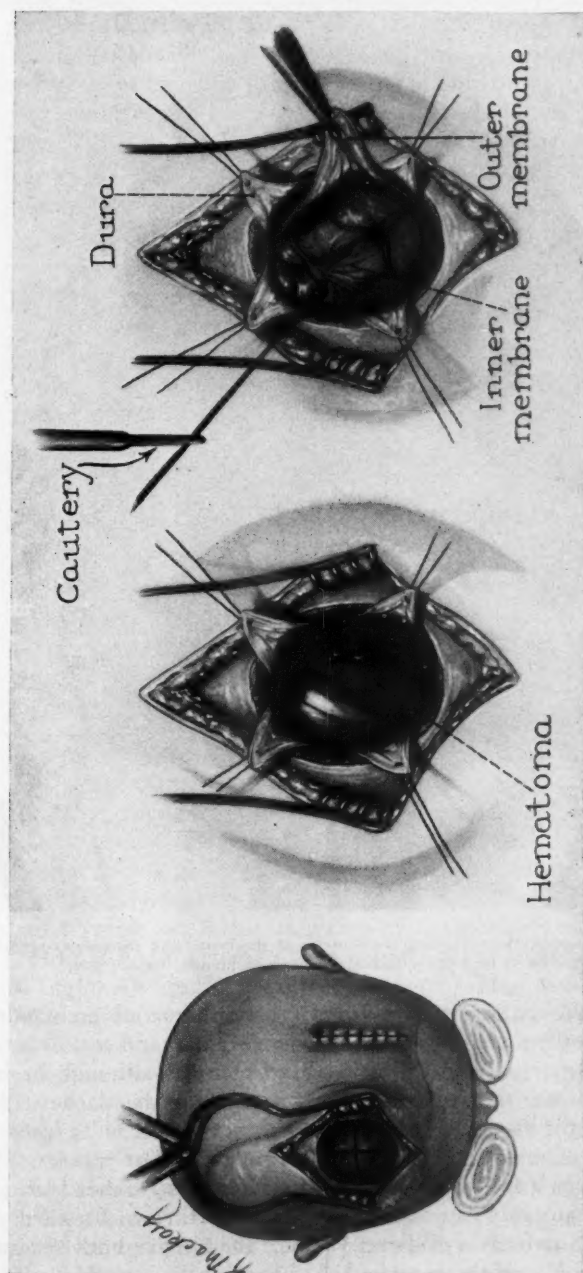
Fig. 2. The trephine openings are those used in carrying out routine ventricular air studies.
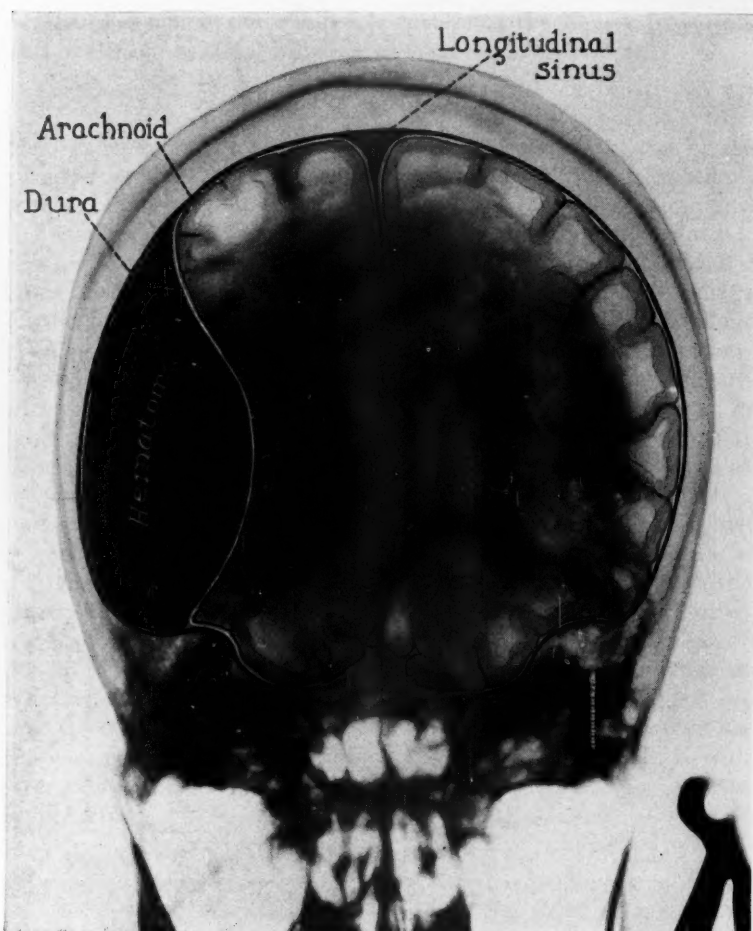
Fig. 3. By thoroughly evacuating the contents of the hematoma sac permits gross destruction of both the subdural and epiarachnoidal membranes.

bruise to his skin resulted in extensive subcutaneous ecchymoses. The other patient was a railroad section foreman and certainly had had many opportunities for trivial head trauma, although he had no specific injury that preceded the onset of his headaches. The character of the blow to the head of those who had definite histories of injuries has been for the most part a polar type of trauma. This has been either a fall with the patient striking his forehead or occiput, or if an automobile accident the head was thrown forward and backward. In two cases of direct violence the blow in both instances was to the middle of the forehead. Although there were histories of

several scalp or forehead abrasions, only in two instances were there lacerations, one of which required suturing. The insignificance of the character of the injury has been noted by almost every contributor on the subject of chronic subdural hematomas and it is only stressed here for the purpose of illustrating the necessity of eliciting a detailed traumatic history in any patient presenting subjective symptoms of any increased intracranial pressure.
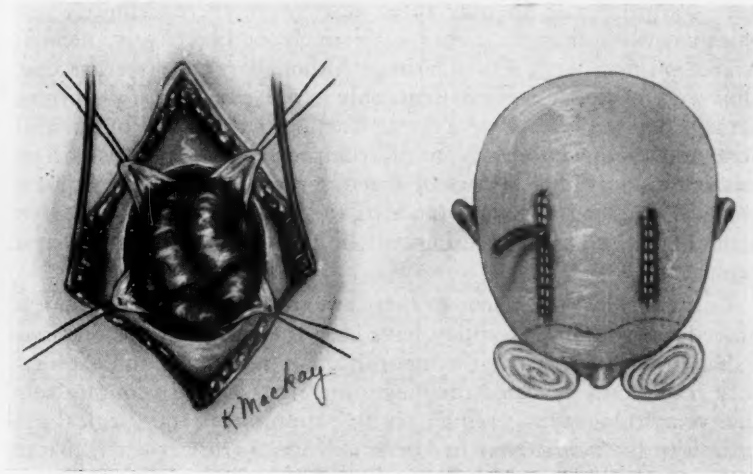

Fig. 4. Before the cerebral tissues have come to full expansion a penrose drain is placed in the depth of the sac.

The most common subjective symptom was headache. In all but the three infants this complaint was the first and most persistent symptom. Although in most instances the headaches were frontal in location there was no localizing value in this symptom. In addition to a frank headache, fourteen of the patients complained of lancet-like "shooting pains" that commonly originated in the temporal region and radiated to the vertex or orbital fossae. In those patients whose consciousness permitted, and whose interpretations seemed correct, at the time of the opening of their duras, it seemed quite definite that the "sharp shooting" pains were reproduced when tension was put upon their duras. Thirteen of the twenty-one adult patients had associated forceful vomiting with their headaches. There was a subjective diminution in visual acuity in eight cases, and only eleven of the twenty-one adults had swelling of the optic nerve heads. In many instances where retinal hemorrhages were absent prior to operation, large flame-like streaks appeared in the retinae on the third to the fifth day after operation. These persisted

in several instances for two to three weeks. Diplopia or blurred accommodation was noted by eighteen patients, seven of whom had unilateral pupillary dilation. In eight cases a definite sixth nerve weakness was demonstrated. Motor symptoms were present in thirteen of the cases and these weaknesses varied from a facial lagging (central type) to almost a complete hemiplegia. As has been pointed out by more than one author, these motor symptoms were more commonly on the same side as the hematoma than they were contralateral. In only three cases were there definitely impaired sensory changes, either subjectively or objectively, demonstrable on neurologic examination. Although routine stereoscopic films were made on every patient, only in one case was any fracture damage depicted. This was a linear fracture in the posterior parietal bone and the hematoma primarily compressed the frontal lobe on this same side. This absence of fracture damage is only of interest in again stressing the importance of the insignificance of the character of the injury that brings about the origin of the subdural hematomas.

In the surgical treatment of these twenty-four cases the technical procedures with one exception have been the same. In this one case a diagnosis of a left frontotemporal tumor was made and exploratory craniotomy revealed the diagnostic error. Since adopting routine ventriculography preliminary to craniotomies the surgical approach to the hematomas had been uniform. The patient is placed in the cerebellar head rest, with slight hyperextension of the head, and under procain hydrochloride anesthesia bilateral trephine openings are made in the frontal bones (fig. 2). These openings are made approximately 5 cm. from the midline and just anterior to the coronal suture. A small opening is made in the dura on both sides. If the hematoma is bilateral the procedure is the same for both sides. If the hematoma is unilateral the scalp is closed on the opposite side and the skull over the hematoma is rongeured away for an area of some 4 cm. in diameter. The dura (fig. 3) is then cruciated and reflected with suture retraction. The subdural membrane (fig. 3) is then incised and the liquid contents of the hematoma sac are thoroughly irrigated and removed with suction. Before damaging the epiarachnoidal membrane, the subdural membrane is teased away as much as possible in bulk and then seared within the subdural cavity by "spraying" with the electrocoagulating current. The epiarachnoidal portion of the hematoma sac is then picked up with the dural hook and widely excised or destroyed. With the patient's head dependent it is most common that the cerebral tissue rapidly pulsate in, to obliterate the subdural space that had been occupied by the hematoma. Before this expansion is complete, (fig. 4) a one-quarter inch penrose wick is fed into the

CASE NO	AGE. SEX	OCCUPATION	TRAUMA HEAD	LENGTH OF TREAT	HEAD-ACHE	VOMIT-ING	DROWSI-NESS	CHOKE DISKS	RETINAL HEMORRH.	CRANIAL NERVES	MOTOR SYMPTOMS	SENSORY SYMPTOMS	PROGRESS. INCREASE	STUPOR COMA	SKULL FRACTURE HEMOR.	SIDE of SKULL HEMORAGE	SURGERY	RESULT
34134	44 M YES	POLICE	AUTO ACCIDENT	1½ MOS.	YES	YES	YES	3D.	YES	NO	YES	NO	YES	NO	NO	LEFT	OSTEO-PLAV	WELL
3614	18 M YES	STUDENT	FOOTBALL INJURY	3 MOS.	YES	YES	NO	3D.	YES	VI RT.	NO	NO	YES	NO	NO	LEFT	TREPHINE DRAINAGE	WELL
3623	63 M YES	TEXTILE	NONE	?	YES	YES	YES	3D.	NO	NO	YES	YES	YES	NO	NO	LEFT	TREPHINE DRAINAGE	DIED.
344	44 M YES	AGENT	AUTO ACCIDENT	3 MOS.	YES	YES	YES	3D.	YES	III RT.	NO	NO	YES	NO	NO	LEFT	TREPHINE DRAINAGE	WELL
36283	62 M YES	PHYSICIAN	AUTO ACCIDENT	2 MOS.	YES	YES	YES	NO	NO	III RT.	YES	NO	YES	NO	NO	RIGHT LEFT	TREPHINE DRAINAGE	WELL
36319	13 M YES	STUDENT	FALL	3 MOS.	YES	YES	NO	2D.	YES	VI LT.	NO	NO	YES	NO	NO	LEFT	TREPHINE DRAINAGE	WELL
5763	31 F YES	H WIFE	FALL	4 MOS.	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	RIGHT	TREPHINE DRAINAGE	WELL
3782	67 M YES	JUDGE	FALL	1½ MOS.	YES	YES	YES	NO	NO	III RT.	YES	NO	YES	YES	NO	RIGHT	TREPHINE DRAINAGE	WELL
37140	51 M YES	CLERK	FALL	3 MOS.	YES	YES	NO	NO	NO	III LT RT.	NO	NO	NO	YES	NO	RIGHT	TREPHINE DRAINAGE	WELL
37180	1 MO.	INFANT	BIRTH?	1 MO	?	YES	NO	NO	NO	VI LT.	YES	NO	YES?	YES	NO	RIGHT	TREPHINE DRAINAGE	RECOVERED
37240	4 MOS.	INFANT	BIRTH?	4 MOS.	?	YES	NO	NO	NO	?	YES	NO?	YES?	NO	NO	RIGHT LEFT	TREPHINE DRAINAGE	RECOVERED
37249	69 M YES	SECRETARY	FALL	3 MOS.	YES	NO	YES	NO	NO	NO	YES	NO	YES	NO	NO	RIGHT	TREPHINE DRAINAGE	WELL
37260	3 MOS.	INFANT	BIRTH?	3 WKS.	?	YES	NO	NO	NO	?	YES	NO?	YES	YES	NO	RIGHT LEFT	TREPHINE DRAINAGE	RECOVERED
31307	47 F YES	H WIFE	FALL	12 MOS.	YES	NO	YES	NO	NO	VI RT.	NO	NO	YES	YES	NO	RIGHT LEFT	TREPHINE DRAINAGE	WELL
37238	64 M YES	ENGINEER	FALL	1 MOS.	YES	NO	NO	NO	NO	III RT.	YES	NO	YES	YES	NO	LEFT	TREPHINE DRAINAGE	WELL
38895	52 M YES	FARMER	AUTO ACCIDENT	1 MO.	YES	YES	NO	NO	YES	VI RT.	YES	NO	YES	YES	NO	RIGHT	TREPHINE DRAINAGE	WELL
391	69 M YES	ENGINEER	AUTO ACCIDENT	4 MOS.	YES	NO	YES	NO	NO	VI LT.	YES	NO	YES	NO	NO	LEFT	VENTRICULAR TREPHINE DRAINAGE	WELL
39939	64 M YES	MERCHANT	DIRECT VIOLENCE	4 MOS.	YES	NO	YES	YES	NO	III RT.	YES	NO	YES	NO	NO	RIGHT LEFT	TREPHINE DRAINAGE	WELL
39277	30 M YES	CIVIL SERVICE	DIRECT VIOLENCE	4 MOS.	YES	YES	YES	3D.	YES	VI LT.	NO	NO	NO	NO	NO	LEFT	TREPHINE DRAINAGE	WELL
30986	64 M YES	STUDENT	FALL	7 MOS.	YES	NO	NO	5D.	YES	III RT.	NO	NO	NO	NO	NO	RIGHT	TREPHINE DRAINAGE	WELL
39606	27 M YES	TEXTILE	CHOKED	3 WKS.	YES	YES	YES	4D.	YES	NO	NO	NO	YES	YES	NO	RIGHT	TREPHINE DRAINAGE	RECOVERED
39507	44 M YES	FARMER	AUTO ACCIDENT	2 WKS.	YES	NO	YES	2D.	NO	?	NO	NO	YES	YES	NO	RIGHT	TREPHINE DRAINAGE	WELL
39548	46 M YES	ALCOHOLIC		2 WKS.	YES	YES	YES	3D.	YES	?	YES	YES	YES	YES	YES	RIGHT LEFT	TREPHINE DRAINAGE	WELL
4084	60 M YES	R.R. EMPLOYEE	NONE.	?	YES	YES	YES	2D.	NO	VI LT.	YES	YES	YES	YES	NO	LEFT	TREPHINE DRAINAGE	WELL
24 CASES	GIVE MALE	ALL TYPES	39 +	FROM 2WK TO 12 MOS. INFANTS	16 +	16 +	11 +	9 +	18 +	13 +	3 +	17 +	12 +	5 +	1 +	8 RIGHT 8 LEFT 8 BILAT	OSTEO-PLAV	1 FATALITY

depth of the cranial cavity and routine scalp closure effected. This drain is removed within twenty-four to thirty-six hours, depending somewhat upon the amount and character of the drainage.

The use of a penrose drain is open to severe criticism. Primarily this is true because of the invitation of an infection into a clean wound. On the other hand, the amount of xanthochromic drainage in most cases is abundant and inasmuch as the absorptive activity of the subdural space is less provided for than even the subarachnoidal space, draining these cases for a few hours has apparently been of help. There have occurred two superficial wound infections in the twenty-three cases, and these two wounds healed promptly by third intention.

In all of the cases treated with trephine drainage the patients are sent back to bed with the head dependent and kept in this position for twenty-four to forty-eight hours. Where there were bilateral hematomas the patient's head is turned from side to side at hourly intervals. This routine position in bed probably is not necessary but when one visualizes the large spaces sometimes remaining after evacuation of the hematomas and realize that this cerebral compression was developed over a period of weeks and in many instances over a period of months, then one could hardly visualize a return of the cerebral tissues to normal bulk within a few minutes. The other reasoning that has prompted the dependent position of the head, aside from the promotion of the drainage of pathologic fluid, is the fact that in many instances a capillary blood supply is usually developed between the dura and its subdural membrane; it has been in the hope that the earlier the cerebral tissues return to their proximity with the dura the more likely this minor pressure might be sufficient to help prevent a secondary clot. These concepts are somewhat theoretical but it is of interest that only in one of the cases has it been necessary to reopen, aspirate or have the patient's convalescence interrupted by any symptoms suggestive of a secondary hemorrhage or a return of their subdural hematomas. This one case was the only fatality in the entire series. This was a case of pituitary basophilism that followed castration in young adulthood and his secondary hemorrhage following operation was as grave externally as it was intracranially.

In summary, twenty-four cases of chronic subdural hematoma are reported. Twenty-three of these cases have been treated with simple trephine exposures with destruction of the hematogenous membranes, and dependent drainage at the time of operation and for twenty-four to forty-eight hours following operation. One death occurred in the series. There were no secondary hemorrhages nor have there been any symptoms of a return of the subdural hema-

tomas in any of the twenty-three cases now after a lapse of one to seven years. It is concluded, that in the highest percentage of chronic subdural hematomas, simpler surgical procedures are preferable to the more formidable craniotomy.

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THE MANAGEMENT OF ACUTE INTESTINAL OBSTRUCTION

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IN THE management of acute intestinal obstruction two considerations are of fundamental importance:

1. The diagnosis:
2. The time element between the diagnosis and the operative intervention.

In no other intra-abdominal condition is the time element—with the possible exception of perforation—of so much importance.

The etiologic factors in the production of acute intestinal obstruction are legion: for practical purposes they are after all relatively few. The diagnosis will comprehend whether the patient has an intact abdomen; in other words, has never been operated upon before the onset of the trouble. Has the patient had previous operations, how many, for what, and how long ago? We may therefore divide the factors that may produce acute intestinal obstruction into the factors of original causation, such as intussusception, herniation, volvulus, foreign bodies, neoplasms, Hirschsprung's disease, non-specific inflammatory changes, stricture and mesenteric vascular occlusion.

Intussusception is pre-eminently a disease of the first two years of childhood, and the description of a crying baby passing blood by rectum, indicates the clinical picture. Intestinal obstruction from herniation, formerly numerically the most frequent etiologic factor, is probably not so today. Volvulus, congenital bands, foreign bodies, are relatively infrequent as the cause of intestinal obstruction in an abdomen that has never been operated upon.

Neoplasms with obstructive symptoms are practically all confined to the large intestine, while non-specific enteritis and stricture are still far from frequent.

Mesenteric vascular occlusion occurs at infrequent intervals and over 60 per cent of them occur in those with marked cardiac disability, and in the aged in which degenerative changes take place. And, finally fecal impaction is a cause to be considered, occurring more frequently in aged females and marasmic children.

Turning to the factors of secondary causation, conditions that arise in an abdomen that has been previously operated upon. We

may divide these factors into those that arise following laparotomies:

1. The immediate postoperative causes of intestinal obstruction:
2. Those that arise as a result of a former operation.

In the immediate postoperative intestinal obstruction the surgeon at least knows the site of the immediately preceding operation. It will be gastroduodenal, small intestine, appendix, pelvic operations such as retroversion, round ligament operations, and hysterectomy, or operations on the large bowel. The mechanism of production will be either bands or plastic adhesions between bowel and an operative surface. It is in the immediate postoperative group that diagnostic difficulties arise, between a paralytic ileus and an incomplete but non-vascular obstruction incident to the development of peritonitis. It is axiomatic that the more numerous the previous operative interventions the more likelihood of intestinal obstruction. A consideration of some of the mechanical and chemical changes that occur in acute intestinal obstruction is indicated in order to comprehend the symptomatology. Anatomically the ileocecal valve may be used as an anatomic indicator. Accordingly, we may speak of an intestinal obstruction of the small intestine as either high or low, and intestinal obstruction of the large bowel. The mechanical effect of an obstruction is to prevent the propulsion onward of the fecal current. The obstruction may be complete or incomplete, and the immediate effect is the regurgitation or back flow of intestinal material from the point of obstruction into the stomach. Vomiting is the inevitable result of small intestinal obstruction. The material regurgitated is at first duodenal contents, then small intestinal contents, changing in color, odor and thickness, and later becoming stercoraceous.

Coincidental or following the mechanical obstruction are vascular changes, first in the blood vessels of the obstructed segment and very shortly changes in the mucous membrane. The intact mucous membrane of the intestine does not absorb toxic by-products but if there is any change in the nourishment of the mucous membrane by strangulation, or thrombosis, or by reason of any arterial or venous occlusive process, then there begins a damage to the mucous membrane which promotes absorption of toxic products and which profoundly alters the entire body chemistry.

The higher the site of the obstruction in the small intestine the more pronounced the symptoms, the greater the degree and more rapid the onset of toxemia, and the greater the mortality. Yet, whether the obstruction is high or low, the cycle of changes above noted are present and the symptoms are fairly constant.

The vomiting or the regurgitation of small intestinal contents is in the nature of a protective mechanism but carries with it pronounced changes in the chemical balance of the blood. First, large and fatal quantities of fluid are lost. The gastro-hepatico-intestinal fluid circulation varies from 7,500 to 10,000 c.c. daily and a patient with intestinal obstruction may lose 4, 6, or even 8 quarts of intestinal contents within 24 hours. Second to the loss of fluids is the loss of hydrochloric acid, hence the peculiar chemical picture of alkalosis, with high carbon dioxide combining power and the development of a vicious circle. Alkalosis increases vomiting, vomiting lessens the acid ions, which in turn increases the alkalosis.

There is a marked renal dysfunction from chemical imbalance and dehydration. The urea-nitrogen of the blood rises and the patient will die with a clinical picture of pronounced intoxication and on autopsy there will be found evidence of hepatic and renal necrosis.

An obstruction of the large bowel will be due ordinarily to volvulus of the sigmoid, extra-luminal tumors, diverticulitis or neoplasms. Five per cent of patients with rectosigmoidal malignancy enter the hospital for acute obstruction. The outstanding clinical feature will be the development of an increasing abdominal distention without vomiting until very late. The continued competency of the ileocecal valve means there will be a delivery of intestinal material into the colon so that one is dealing practically with obstruction in a closed loop. Here the distention becomes so great that the intraluminal pressure blocks the venous exit of blood and thereafter prevents the inflow of arterial blood. Thus is initiated a mechanism for widespread necrosis of the bowel but there may be one or two bowel movements below the obstruction after which obstipation is the rule.

In acute intestinal obstruction in the small bowel the initiatory symptom is pain which comes on suddenly, is paroxysmal in type, is associated with restlessness, is controlled with difficulty by narcotics, and simultaneously or shortly after the onset of pain there is vomiting. The vomiting is repetitive, without effort, and the character of the material changes rapidly. The abdomen is, as a rule, not distended but is everywhere somewhat sensitive. Plastic operations on the pylorus, without gastroenterostomy, may obstruct by angulation, kinking, and plastic exudate. Posterior gastroenterostomy or a Polya operation may be associated with retraction of the stomach upward through the mesocolon and thereby produce acute angulation of the jejunum with obstruction. This has occurred four times in my practice and I have had to relieve an herniation of the small intestine through a defect in the mesocolon sixteen years after a

gastroenterostomy. Plastics on the pylorus have, in my opinion, been unsatisfactory on account of the possibility of postoperative intestinal obstruction.

One must remember that the midportion of the small intestine is under ordinary circumstances resting in the cul-de-sac of Douglas, and pelvic operations are associated with the possibility of adhesions of small intestine to the under surface of an hysterectomy wound or to other portions of the pelvis. Again, any portion of the small intestines may become attached to an operative area, as a resection of Meckel's diverticulum was followed by an intestinal obstruction. Not infrequently after a simple appendectomy there is an adherence of the bowel to the appendiceal wound. This may occur during the course of ordinary convalescence, or again it may occur any time after the appendectomy.

When we survey the intra-abdominal inflammatory diseases we may say that of all the factors producing acute intestinal obstruction, infection is pre-eminent in frequency. Numerically acute intestinal obstruction following acute appendicitis heads the list in frequency. The surgeon many times in operating for acute appendicitis is certain that the inflammatory changes in the terminal ileum are so severe as inevitably to produce obstruction. It has been our practice to counteract this by passing a tube through the stump of the appendix into the terminal ileum, or to perform a terminal ileostomy at the time of the operation for appendicitis.

The institution of intra-abdominal drainage permits only the drainage of free flowing fluid material. It does not operate like a sewerage pipe. The outstanding usefulness of drainage is to prevent the products of infection from being retained under pressure. The greatest worry or concern of the surgeon in acute appendicitis is not the inflammation of the appendix but whether the condition present will lead to a progressive diffusing peritonitis. A diffuse peritonitis begins and ends as an intestinal obstruction, for the bowel in proximity to the infectious area develops first a serositis, characterized by transudation, followed by exudation; the wall of the bowel is involved, becomes red, turgid and obstructed, and we have then all of the mechanical and vascular conditions that pertain to a small intestinal obstruction. A bowel in a condition of acute inflammation does not permit the onward propulsion of its material, whereas grave defects in a non-inflamed bowel will, many times with practically no symptoms, permit intestinal movement with propulsion of the intestinal contents.

An acutely inflamed appendix very quickly involves the terminal small intestine and soon the loop of sigmoid becomes a participant in the same process and we have again the same changes taking

place. Thus is added to a primary small intestinal obstruction a secondary sigmoidal obstruction—an ileus duplex. The patient has symptoms of regurgitation from a small intestinal obstruction and also the closed loop distention of large bowel obstruction. For some period of time the bowel above the obstruction, either large or small, participates in its normal contractibility, or exaggerated contractibility and only very late does it become paralyzed as is evidenced by the continuous reflux and purposeless vomiting. The peristaltic and colicky pain gives place to chronic pain and with localized tenderness in the neighborhood of the obstruction. A dynamic ileus or paralytic ileus, as a rule, is devoid of the vomiting phase of the mechanical obstruction and the distention is marked, yet upon using a stethoscope diminished or muted peristaltic sounds may be heard showing that while there is an atonicity of bowel it is still a bowel with some peristaltic movements. In the mechanical obstruction these sounds are exaggerated to a more or less continuous loud, gurgling sound and as the bowel dies we hear sounds like a bell jingling as fluid moves around in a distended closed loop, and finally in the moribund stage of peritonitis a completely silent abdomen—a prelude to death.

The management of any condition suspected of being an acute intestinal obstruction divides itself into three procedures:

1. Chemical or replacement therapy.
2. Mechanical therapy and drainage procedures.
3. Surgical intervention.

Under chemical and replacement therapy, fluid lost from the stomach must be replaced volume for volume by water plus the estimated 1500 c.c. of water loss by the skin, plus the 500 c.c. of respiration loss and the minimal 500 c.c. to maintain kidney water. The chlorides must be replaced in sufficient amount to bring the chloride content up to the normal of 450 mg. per 100 c.c. A chemical examination of the blood which shows a loss of chlorides below 400 mg. means widespread chemical imbalance. Chlorides must be given only in sufficient amount to replace the depleted blood chlorides, for an excess very definitely brings about a diminution in the plasma protein concentration from the normal level of 7.5 Gm. per 100 c.c. of blood, to a critical level of 5.5 Gm. below which point tissue edema ensues. Such edema is not confined to the extremities but will occur in the chest, liver and all of the intra-abdominal viscera and a vicious circle may thereby be set up producing in itself the symptomatology that will closely simulate acute intestinal obstruction. Body nutrition may and can be maintained by blood transfusions.

Mechanical therapy will embrace the measures that are habitually employed for diffuse peritonitis. The Fowler position, absolutely nothing by mouth, and such narcotics as may be necessary to allay pain and apprehension. Both vomiting and distention are relieved by a nasal indwelling catheter of the Jutte or Levin type, or a Miller-Abbott tube, or employing Wangensteen's continuous suction apparatus. It is, however, possible by means of such procedures to make a patient so comfortable as to cloak the menace of an impending death, or to drain an excessive amount of electrolytes from the upper gastrointestinal segment and thereby add to the already dangerous condition.

Surgical intervention should be done early. It is better to operate and not find any intestinal obstruction than to delay and demonstrate one. If the diagnosis of acute intestinal obstruction is entertained, operative intervention is indicated before either the chemical imbalance or the distention becomes marked. In the immediate postoperative intestinal obstruction, a band or an adhesion, or a kink, or an angulation is easily corrected, particularly if one remembers to look for obstruction by handling the collapsed bowel and not the distended bowel. I personally have not believed that mortality was increased by evisceration of the patient providing the intestines are kept properly protected. A great deal of controversy hinges upon enterostomy. I cannot recall that enterostomy in my hands has been life-saving. It is perhaps because it was done too late on moribund patients, and if one reads the protocols of enterostomized patients he will read "enterostomy tube drained 150 c.c." and then "drained some more," and finally "no drainage from tube." Treves in 1884 deprecated against it as "a procedure of wasted opportunity," temporarily beneficial but it did not meet the condition which was obstruction.

My results with the Handley operation in general peritonitis have been satisfactory and I would not hesitate to employ it much earlier than heretofore. At the same time one must admit that one of the sequela that may be anticipated is that the jejuno-colostomy must be closed at a later operation.

CYSTS OF THE VAGINA
With Report of a Case

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ALTHOUGH accorded little attention in medical literature, cysts of the vagina are believed to be of more common occurrence than was formerly supposed. Certain it is that cysts larger than an almond are seldom encountered. Some gynecologists believe that many vaginal cysts, being asymptomatic, remain undiscovered, until examination for some other condition draws attention to them.

Vaginal growths of this type were observed by Oakley Heming as early as 1830. Stokes in 1898, reporting on the etiology and structure of cysts of the vagina, found 11 cases in only two of which the cyst was larger than an egg, in a series of 5,000 of Dr. Howard Kelly's gynecologic cases in Johns Hopkins Hospital. Cullen in a subsequent communication from the same hospital, reported 53 cases occurring prior to 1904. Stokes and Cullen summarized in detail what was known of this subject to 1905, and little new has been added to our knowledge since that time.

Von Preuschen in 1877, demonstrated the presence of true vaginal glands in 4 out of 36 bodies. He described the necks of these glands as being lined with squamous epithelium, while the deeper portions, which spread out into definite bays, were lined with cylindrical epithelium on which the cilia might be detected; small crypts opened into the dilated glands. Heming, in 1870, had described similar glands.

Factors which are considered of etiologic significance in the production of vaginal cysts are,—(1) embryonic structures which may persist in the vagina; (2) changes in the vagina incident to vaginitis; (3) changes due to injuries resulting from childbirth.

The persistence of several types of embryonic structure has been demonstrated. Remnants of Gartner's duct have been found in a number of cases in conjunction with existence of a vaginal cyst. In some instances cysts have occurred when Muller's ducts have failed to unite, resulting in the formation of one fairly well developed uterus, and a second represented only by a rudimentary organ or a cord. The walls of the two vaginas may here become fused, and secretion, accumulating in the rudimentary organ, may lead to the formation of a cystic tumor. Likewise abnormalities in the ureter and its position in relation to the vagina may occur with a fistulous opening and constant discharge of urine into the lateral vaginal wall.

The second etiologic factor noted above is found in cases of severe vaginitis where, due to inflammatory processes, the vaginal folds become adherent to one another and blind pockets are produced. This condition may be caused by gangrene resulting from a foreign body in the vagina, to some severe form of general systemic infection, or to the irritative action of urine in cases of severe inflammation of the bladder.

Injuries resulting from childbirth, such as tears in the mucosa within the outlet, or lacerations of the perineum, in healing may likewise form pockets, especially adaptable for the formation of vaginal cysts. Bits of squamous epithelium may in these cases remain buried from 1 to 5 mm. beneath the surface of the perineum, in either slight or extensive lacerations, resulting in time in a closed pocket and a cyst of this type.

In 52 cases reported by Dr. Cullen, 26 were the result of a perineal tear or a perineal repair; 4 seemed to originate from vaginal glands; 3 were located near the external orifice of the urethra; and in 8 it was impossible to determine the etiologic factors.

Since most vaginal cysts are small, they usually cause no obstruction and often produce no symptoms at all. If they attain the size of 6 to 8 cm. in diameter, the vagina may be partially blocked. Occurring in the posterior wall, the appearance may at times lead one to diagnose prolapsus of the vagina. In the author's case, owing to the growth and size of the mass, pain in the left inguinal region was reported as of three years' duration.

Vaginal cysts of small size should be removed, and the raw surface edges should be sutured to leave a normal vaginal wall. Large cysts which cannot be treated in this manner may be incised, removing and suturing the edges at a later date when the vaginal wall has assumed a more normal condition.

REPORT OF CASE

A single, white woman, 37 years old, was first seen on Dec. 17, 1940. Menstrual history was normal, but the patient had suffered moderate pain in the left inguinal region for three years. Palpation of the left lower abdomen was painful.

Induration and increase in the size of the left fallopian tube was noticeable on bimanual examination of the pelvis. A cyst of the left ovary, about the size of a hen's egg was discovered, in addition to a rather firm tumor in the floor of the vagina about midway between the introitus and the cervix. This mass was oblong in shape, and measured about 6 by 3 by 3.5 cm. Digital examination showed the tumor not to be connected with the rectum, but to lie just within the posterior vaginal wall. Owing to the size of the mass, it was difficult to pass a bivalve speculum beyond it. The surface of the vaginal wall over the cyst showed only normal epithelium, without ulceration or discoloration.

At operation the left fallopian tube and the cyst of the left ovary were removed, following which the walls of the vaginal cyst were excised as close to the rectovaginal wall as possible. The remaining portion of the cyst wall was curetted gently and its edges sutured. Several examinations within the following two months have revealed a normal posterior vaginal wall.

The cyst on removal showed no capsule, and contained a thick yellowish fluid resembling infected mucous material.

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MANAGEMENT OF ABORTION

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A S TAUSSIG[1] so aptly states, "we hear much about the conservation of our natural resources, reforestation, soil preservation and control of damage by flood and famine. Not so much thought is given, however, to the even more important conservation of our human resources. It is up to us as physicians to take the leadership in that direction. I know of no more glaring example of the failure to practice conservation than in the wastage of human life associated with spontaneous and induced abortion." The same author[1, 2, 3] estimates that 600,000 to 700,000 abortions occur annually in the United States and of this number, 30 per cent are spontaneous. Even more important are his conclusions that spontaneous abortions occur in 10 per cent of pregnancies and that almost all occur within the first twelve weeks of pregnancy.

In this era where voluntary control of conception is widespread, the alarming aspects of his statistics loom as a distinct challenge to the gynecologist and obstetrician, not only as regards the importance of lowering maternal and neo-natal deaths, but in carrying pregnancies to viability and term that otherwise might be lost.

THREATENED AND HABITUAL ABORTION

Though retrodisplacements, deep lacerations of the cervix, fibroids, chronic nephritis, syphilis and focal infections (Curtis[4], Reith[5], Paul and Galloway[47]) account for a small percentage of spontaneous abortions, the larger number of them are due to defective germ plasm, defective or abnormal implantation of the ovum, endocrine imbalance or dietary deficiency in the mother; the latter factors being closely interrelated. The anterior pituitary-like hormone, progesterone, thyroid extract and vitamin E have all been used in an attempt to combat these deficiencies.

Mall[6] is of the opinion that pathologic embryos are developed from normal ova and are due to external or environmental influences. Malpas[7] is of the same opinion. Recently Greenhill[8], in a study of 4,446 cases of placenta praevia, found the incidence of

fetal monsters and deformities to be 2.5 per cent as contrasted with .94 per cent for all obstetric cases. This conclusion added to Mall's observation that 96 per cent of ova obtained from tubal pregnancies were abnormal, lends weighty evidence to the fact that not only is a normal well differentiated endometrium necessary for the growth and viability of the ovum, but is also necessary for the normal development of the fetus. Certainly, the majority of embryologists today believe that though the tendency to produce fetal monsters and deformities may be inherited and in the germ plasm, the larger number of fertilized ova are normal and fetal deformities result from environmental causes and are developmental origin. Should, then, one be able to arrest spontaneous abortion and carry the pregnancy to term, does he not run a great risk of delivering a monster or a deformed child? That this risk is more theoretical than real is shown by Shute[9] who has collected the data in the literature on fetuses delivered after threatened abortion had been successfully controlled by means of vitamin E or progesterone or both, and found that of 443 cases so treated, only 2 per cent expelled a deformed fetus, concluding "that vitamin E and progesterone may actually facilitate a completion of the inadequate development of certain types of defectives if given in time."

Gonadotropic and Corpus Luteum Hormones

The hypophyseal sex hormones, prolan A and B are necessary for the proper development of the follicle and corpus luteum. A well functioning corpus luteum is necessary for the progestional or well differentiated pregravid endometrium which in turn is so important in the proper embedding of the ovum, its nourishment, and the subsequent formation of the placenta. Until the placenta fully forms, excision of the corpus luteum is usually followed by abortion. However, following development of the placenta, the corpus luteum is no longer necessary for the preservation of pregnancy, as the placenta elaborates estrogens, anterior pituitary-like hormones and progesterone[10]. There is very little biologic difference between the pituitary and placental gonadotropic factors, especially the luteinizing fraction and the latter has been successfully used in the treatment of threatened abortion.

Wiesner[11] observed that a negative Aschheim-Zondek reaction in a living pregnancy implied a high risk of abortion or premature birth. Robson[12] and Crew[13] share the same view. Potter[14,15] in her studies proved that there is no connection betwen the amount of prolan A excreted by a pregnant woman and the continuance of pregnancy to term, but that the Aschheim-Zondek reactions were definitely weaker in a series of patients who habitually aborted than

in a similar series performed on normal pregnant patients, pointing to a lack of the B or luteinizing factor.

Sellheim[16] was the first author to report the use of normal pregnancy serum in the treatment of habitual abortion, successfully carrying eight out of nine patients to term. Rosenfeld[17] treated twenty cases of habitual abortion using the same therapy and nineteen gave birth to normal living infants. Gershenfeld[18] treated sixteen cases with antuitrin-S and carried twelve to term. Whether success was accomplished as a result of mere substitution of hormone or hormones deficient in the pregnant women or whether the anterior pituitary-like hormones stimulated corpus luteum formation and consequent elaboration of progesterone is a matter of conjecture.

Corner and Allen[19] have shown that the corpus luteum hormone, progesterone, is essential for the maintenance of early pregnancy. The properties of progesterone to relax uterine musculature (Falls, Lackner and Krohn[20]) and, following estrogenic stimulation, produce a well differentiated type of endometrium in the uterus have been well demonstrated (Weichert[21], Corner and Allen[22]). Krohn, Fall and Lackner[23] believe that in spontaneous abortion "there is a deficiency of the corpus luteum hormone, either relative or absolute, which may manifest itself in several ways; (1) By an excess and overactivity of estrin due to diminished excretion which sensitizes the uterus to the action of pituitrin, thus initiating contractions, (2) By inadequate decidual reaction which may result in (a) undernourishment and death of the fertilized ovum after implantation with automatic expulsion and absorption, (b) deficient placentation which in turn may cause a deficiency of progestin in the placenta, and (c) by increasing the hemorrhagic tendency in uterine endometrium produce premature detachment of the placenta in certain cases." Based upon this logic the same group of authors[20] treated forty-one cases of threatened or habitual abortion with progesterone and obtained successful results in thirty-four patients. In ninety-four previous pregnancies in this same group, sixty-five spontaneous abortions had occurred. Elden[24] and Kane[25] have also reported good results using the corpus luteum hormone. Krohn and Harris[48] have recently reported excellent results using pregnaninolene orally (anhydrohydroxy-progesterone).

THYROID EXTRACT

Litzenberg[26, 27, 28] has shown the close relationship between the thyroid gland and sterility and abortion. He reports 33.3 per cent conception with only 14 per cent abortions in a series of fifty women. King and Herring[29] are convinced that hypothyroidism plays a large role in spontaneous abortion and also in cases of "missed abortion."

The exact relationship of the thyroid gland to spontaneous abortion is as yet unsolved. It is well established, however, that the thyroid is definitely interrelated with the other glands of internal secretion. Haines and Mussey[30], Novak[31] and others believing that thyroid extract is the most useful of all glandular preparations in cases of amenorrhea and sterility. With this opinion we heartily agree.

Wheat Germ Oil (Vitamin E)

In 1921, Evans and Bishop[32] reported that rats on supposedly normal diets, though growing normally and exhibiting normal estrous cycles, would breed and conceive but fail to deliver because of death and resorbtion of the fetuses. When whole wheat cereal, fresh lettuce leaves or dried alfalfa were added to the diet normal reproduction occurred. Sure[33] working along similar lines arrived at the same conclusion and proposed the name "Vitamin E" for the new antisterility factor. Rowlands and Singer[34] conclude that vitamin E deficiency causes a definite decrease in the capacity of the non-pregnant rat to cause ovulation in the estrous rabbit, and therefore, presumably a decrease in the content of the luteinizing or ovulation producing substances. The same authors and Daikov and Krizenecky[35] (quoted by Rowlands and Singer[34]) have shown that the administration of anterior pituitary or pregnancy urine extracts does not prevent the characteristic resorbtion of the fetuses or vitamin E deficient rate. Shute[36, 37] believes that the beneficial results of vitamin E are due to its neutralization of all antiproteolytic ferment found in the blood of women who habitually abort and that vitamin E is anti-estrogenic. Of much significance is the work of Evans and Burr[38] who reported that in vitamin E deficient rats there is pathologic development of the embryo and embryonic contributions of the placenta but no pathology in the uterus or endometrium. Furthermore, they found that these changes prevented successful implantation of the ovum and that vitamin E in adequate dosage, administered even as late as a few hours before implantation, resulted in normal implantation and gestation.

These observations lead to but one conclusion, namely, that though the exact mode of action of vitamin E is unknown, it is necessary in the maintenance of normal gestation. Vogt-Muller[39] first reported on the use of vitamin E in habitual abortion. Since then, numerous reports have appeared in the literature. Watson[40] reporting successful results in forty out of fifty-two women who had had habitual or threatened abortions. Bishop[41], Croemer[42], Currie[43] and Watson and Tew[44] all report excellent results using the same mode of therapy.

In a previous report, we have summarized our results obtained by using a combination of thyroid extract, vitamin E and progesterone in the treatment of 24 cases of threatened abortion and 12 cases of habitual abortion[49]. We are now able to report a total of 61 consecutive cases of threatened abortion and 17 consecutive cases of habitual abortion occurring in our private practices. These are not selected cases, but represent cases having either been in our care when the abortion began or presenting a threatened spontaneous abortion when first seen.

Up until six months ago, when synthetic vitamin E became available, we used cold pressed wheat germ oil. Since the availability of synthetic vitamin E, we have used this product with equally good results; the synthetic product is more stable and easier to administer. Aside from this minor modification, the mode of therapy has been the same as described in our earlier series.

When using the cold pressed wheat germ oil, we never prescribed more than 2 ounces at a time, and the patient was advised to keep the oil in the refrigerator because of the ease with which it deteriorated[45]. The daily maintenance dose ranged from 1 to 1½ drams unless the patient began to show signs of threatened abortion when first seen. Eight to twelve drams were given during the first twenty-four hours to these patients as advised by Shute[46], and then the patients were placed on a daily maintenance dose. The oil was given until the patient reached eight to eight and one-half months of pregnancy. Since using the synthetic vitamin E (14 cases), we have prescribed 10 mg. twice daily when the patient was showing signs of abortion, and once the abortion had been quieted, the daily maintenance dose was reduced to 6 mg. per day until the patient reached 8½ calendar months. For cases of habitual abortion, 6 mg. daily was prescribed.

One rabbit unit of progesterone was administered daily, intramuscularly, when cramping or bleeding was actually present. Upon cessation of these symptoms, the corpus luteum hormone was then given once weekly until the time of placentation was complete, usually 4½ months. However, in cases where uterine cramps or bleeding was observed following discontinuance of corpus luteum therapy, progesterone was given once weekly until the patient reached 8½ calendar months of pregnancy. During the past two months, we have used anhydrohydroxy-progesterone (pranone), orally, in the place of hypodermic administration of progestin and it is our impression that this method of administration is as efficacious as the hypodermic administration of progestin. Although we have not used it in enough cases to establish firmly a view one way or the other, Krohn and Harris[48] have shown recently that good results

may be expected from this mode of therapy in cases of spontaneous or habitual abortion. They treated 50 consecutive of threatened habitual abortion, and report that in 39 cases of habitual abortion—success was attained in 32 (82 per cent) and failure in 7. Patients with threatened abortion received 10 mg. of pranone twice daily until all symptoms subsided, and then took 10 mg. twice weekly for several months. Patients with habitual abortion received 10 mg. twice weekly for several months.

Thyroid extract remains a basis of our therapy and was given to all patients who showed evidence of hypothyroidism, such as low B.M.R., obesity, irregular menstrual periods, and slow pulse. The dosage ranging from 1/10 of a grain to 2 grains daily, depending on the patient's tolerance to the drug. Under this therapeutic regime, our results have been as follows:

THREATENED ABORTION

No. of cases..61
No. of cases successfully combated........................50 (82%)
Failure 11 (18%)

HABITUAL ABORTION

No. of cases..17
No. of pregnancies before therapy........................31
(3 live births)
No. of pregnancies with therapy..........................15
(15 successful—79%)

The percentage of results obtained in the series of 78 cases closely approximates the same percentages obtained when our series contained only 38 cases, there being little or no variance. The fact that we have obtained fairly consistent results using this mode of therapy lowers the chance of statistical error. Of all the children delivered from this group, only one has shown any evidence of deformity, and that was a case of mild hypospadius, which deformity is so slight that surgical correction will not be necessary. Recent work with vitamin C impresses one with the fact that this vitamin is also necessary for a proper continuance and maintenance of the fetus in utero. Routinely, our antenatal cases are placed on large amounts of citrus fruit and at least two lemons a day; as a result, none of our patients showed any deficiency in this vitamin. However, it is well to keep in mind the experimental evidence that vitamin C deficiency does play a part in habitual abortion. The ease with which this vitamin is obtained from citrus fruit makes substitutional therapy unnecessary except in cases where persistent nausea and vomiting complicates the picture.

MISSED ABORTION

Occasionally a fetus will die in utero and will not be expelled. When such is the case, a condition of missed abortion is present. Many authors differ in their definition of missed abortion, but the majority believe that should the fetus be retained longer than three to four weeks, the case is one of true missed abortion.

The diagnosis of missed abortion is made by the observation that the size of the uterus is not compatible with the period of amenorrhea; that there is no further growth in the size of the uterus; that there is a cessation of fetal heart tones. If the pregnancy be passed five months, x-ray examination will show the characteristic signs of fetal death, namely, overlapping of the skull bones, asymmetry of the head, collapse of the thoracic cavity and horseshoe curvature of the spine, (Matthews[50]; Stein[51]). When the pregnancy is too young for osseus tissue to be demonstrable by x-ray, then biologic tests may be of value. Rezek[52] claims that accuracy of 95.3 per cent in a series of 86 cases using a modification of the Schnieder[53] test. Spielman, Goldberger and Frank[54] showed that with intrauterine death of the fetus, there is a fall in the amount of free estrogens in the blood stream and suggested that if less than one mouse unit of estrogen is demonstrable in 40 c.c. of blood of a pregnant woman, the finding is diagnostic of intrauterine death. This work was confirmed by Jeffcoate[55].

Once the diagnosis of intrauterine death of the fetus has been made, the problem arises as to what mode of therapy one shall use to empty the uterus. We have adopted a very conservative attitude, and it is our policy to watch the patients carefully and interfere only if untoward signs or symptoms developed. The majority of cases of missed abortion will be expelled from the uterus after some time without any interference whatsoever, and it has been our experience that any interference adds great risk of hemorrhage and subsequent infection. Therefore, we have limited our therapeutic measures to simple medical induction using castor oil and pituitrin, repeatedly if necessary. Should these simple measures fail and the patient show untoward signs to symptoms, then, and only then, do we resort to surgical interference. Should the necessity for surgical interference arise, catheters inserted into the cervix, or packing the cervix by means of gauze, might help in initiating contractions. Occasionally, vaginal or abdominal hysterotomy are used, and in very rare cases, abdominal hysterectomy may be necessary.

In 1935, Robinson[56] reported a new method for evacuating the uterus in missed abortion and missed labor by increasing the tonicity of the uterus with estrogens. Later, Jeffcoate reported his experience using this method of treatment in 55 consecutive cases. Estra-

diol benzoate was given intramuscularly in 2 mg. doses every 8
hours for seven to eight days. If abortion did not take place on the
fifth day, quinine hydrochloride, grains 10, was given each hour for
3 doses and followed by 4 injections of 0.5 c.c. of posterior pituitary
extract given at intervals of one hour. This treatment was repeated
on the eighth day if the product of conception still remained in the
uterus. When stilbestrol was used instead of estradiol benzoate, 2
mg. was given by mouth three times daily or 1 mg. every four hours
for seven to eight days. The quinine and pituitary extract were also
given when required on the fifth and eighth day. Using this method
the author reports seven failures, but stated that failures were
fewer once experience with the method was acquired. Thus, there
was only one failure in the last twenty-seven cases. Every patient
successfully treated with the estrogenic hormone had an uneventful
apyrexial convalescence, and the extrusion of the uterine content
was usually rapid, complete and unaccompanied by hemorrhages. In
one case, the delivery of the placenta was delayed for a few hours.
In the past two years, we have attempted to empty the uterus, in
cases of missed abortion, by means of estrogens, using the synthetic
product stilbestrol, administering 5 mg. three times daily, for five
days, and have been unsuccessful in 4 cases. However, we must
admit that our method of induction was not exactly the same as that
followed by Jeffcoate[55], and in the future, we will attempt to use his
method and see whether or not we can better our results. King[57], of
the Department of Obstetrics at Tulane University, states that he
has used stilbestrol in two cases of missed abortion, and that it is
his opinion that the estrogen aided in emptying the uterus.

INCOMPLETE AND SEPTIC ABORTION

If the patient gives a history of induced abortion or if attempts
to arrest a spontaneous abortion fail or should the case be one of
incomplete abortion when first seen, therapy should be directed
towards the prevention not only of blood loss, but also of the intro-
duction of or spread of infection. Here as with cases of missed
abortion we adopt a policy of utmost conservatism.

The patient is immediately given 1 c.c. of pituitrin, intramuscu-
larly, and examined vaginally under sterile precautions. The
amount of dilatation of the cervix is noted, and should fetal prod-
ucts be presenting at the os, they are removed by means of a sponge
holder placed at the external os, but not into the cavity of the uterus.
Under no circumstances do we invade the cavity of the uterus
either with the sponge forceps, the dull curette or the finger in an at-
tempt to remove the products of pregnancy, whether or not the pa-
tient gives a history of an induced abortion or whether or not she

has temperature when first seen. Oxytoxics are then given, prefer-ably ergotrate, 1/320 grain every 6 hours for eight doses. Should the administration of oxytoxics and removal of whatever products of pregnancy are presenting at the external os fail to control the bleeding, the vagina is packed. The pack is not inserted into the uterus or into the cervix but is placed tightly against the cervix and the vagina is packed to capacity. This is best accomplished with the patient in the Sims' position, and one should make certain to re-move all of the blood clot from the vagina before inserting the pack. The pack is left in for 24 hours, and then removed. Should the patient cease to bleed, no further packing is required. However, should the patient bleed following removal of the pack, the cervix is then inspected by means of a speculum and any products of preg-nancy presenting at the os are then removed. Should no products of pregnancy be found presenting at the os, and should the patient con-tinue to bleed, or should the patient continue to bleed following the removal of products of pregnancy, the vagina is again repacked for a period of 24 hours.

Under this regime we have been able to control all bleeding aris-ing from incomplete or septic abortion and have not had to perform an immediate curettage, or empty the uterus by means of the sponge holder inserted into the cavity of the uterus, for the past five years. As a matter of fact, it is very rare that the combination of oxy-toxics and removal of whatever products of pregnancy present at the external os, do not suffice to control the bleeding; even the ap-plication of vaginal packs have been quite rare on our service.

Under no circumstances, do we invade the cavity of the uterus, unless the patient has been afebrile for a period of 7 to 10 days. Should the patient fail to cease bleeding 7 to 10 days after admis-sion and should her temperature be normal for 7 to 10 days, then, and only then, will we curette a uterus. Though some authors be-lieve that the patient's hospital stay and convalescence will be shortened, and the increase in mortality and morbidity rates be but slightly raised by means of early curetment, we do not believe that we are justified in taking these risks no matter how slight the in-crease in mortality might be. Witherspoon[58] in 1933 reported on 200 cases of septic abortion treated on the wards of Charity Hos-pital. These cases were representative of those treated on the Tu-lane Unit where the utmost conservatism prevails, as compared with a similar number treated on other units at that time where operative interference prevailed. He found that in 100 cases which were treat-ed by some operative procedure, the minimum of which was dila-tation of the cervix and curetment of the uterus, the mortality was 9 per cent, whereas, in the second 100 cases treated only with seda-

tives, rest and other conservative means, there was a zero per cent mortality. Sixty-four patients whose uteri were curetted were made more septic by the operation, as judged by increase febrile reaction, and consequently, their convalescence was made more stormy.

Should the patient be febrile on admission, and the temperature be under 102 degrees F., no further therapy is given until the patient has been observed for 48 hours, as most of these patients show a tendency for the temperature to drop under the conservative method of therapy. Should the patient's temperature be over 102 degrees F. on admission or should the temperature show no tendency to drop in 48 hours, then the patient is placed on chemotherapy for 72 hours. We have used sulfanilamide and more recently sulfathiazole as chemotherapeutic agents and prefer the latter drug; we might state also that we do not wait for cultures, either blood or uterine before administering chemotherapy to septic cases. Our routine is 4 grams of sulfathiazole immediately, and 2 grams every 6 hours for 48 hours and we have been impressed by the quick subsidence of fever in septic cases using this mode of therapy. Should the patient be anemic, 500 c.c. of blood transfusion are given and re-peated every 2 to 3 days in smaller doses, 250 c.c., until the patient is no longer septic. Should the patient on admission show evidence of pelvic peritonitis, then the patient is treated by means of Wangen-steen suction tube, nothing by mouth; fluids administered intra-venously, 2000 to 3000 c.c. a day; blood transfusions given as de-scribed previously, and sulfanilamide in the form of clysis, 250 c.c. of an 0.85 per cent solution, freshly prepared, twice daily until all evidence of peritonitis subsides.

Should a patient continue to run a septic course, her pelvis is checked at weekly intervals, in an attempt to determine whether or not the infection is localizing as an abscess of the cul-de-sac, para-metrium or adnexae. Should abscesses form in the cul-de-sac, they are drained through the posterior fornix of the vagina. Should they develop in the parametria and point vaginally, they are drained through the right or left fornix. Should, however, the infection point in the inguinal region and the abscesses are not accessible by the vaginal approach, they are drained by extraperitoneal incision in the region of the Poupart's ligaments.

For the period beginning Jan. 1, 1939 and ending Jan. 1, 1941, 718 cases of incomplete or septic abortion have been admitted to the wards of the Gynecological Unit at Charity Hospital. Of this number, 9 died, and only 48 were curetted. A mortality of 1.25 per cent. These figures represent consecutive cases and are not corrected mortality statistics. Dr. C. Gordon Johnson of our staff is now preparing a complete resumé of all the cases of incomplete or septic

abortion entering our service during the past 10 years. Time and space did not allow for such a presentation in this paper.

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MESENTERIC TUMORS

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A DISCUSSION of mesenteric tumors is probably more interesting than practical in that the treatment of most abdominal masses is usually rather obvious. However, the satisfaction of arriving at a correct diagnosis in an obscure case well justifies our consideration of these lesions.

Secondary tumors of the mesentery are common. The most frequently seen are metastatic carcinomatous nodules following other intra-abdominal malignancies. Less frequently observed are enlarged mesenteric glands representing only a local manifestation of a rather generalized Hodgkin's disease, tuberculosis, lymphathic leukemia, or syphilis. Entirely different and even less frequent are the implants of pseudomyxoma peritonei. We have had one such case which has been previously reported. Since all of these may be generally considered as accidental developments from a primary source elsewhere, their diagnosis treatment and prognosis are more properly a part of the consideration of the original disease.

In contrast to secondary tumors, primary tumors of the mesentery are rare. The least frequent varieties include primary connective tissue tumors of the mesenteric blood vessels, fibrous tissue tumors, sarcomas, lymphosarcomas, and lipomas. The most common type is some form of cyst. Some of the more rarely seen cysts are (1) hydatids, (2) dermoids, usually teratomatous and retroperitoneal; (3) gas cysts, which are small and multiple and whose contents are odorless since the gas is derived from cell activity or invading bacteria and not from the intestinal tract; (4) embryonal rests or fetal inclusions, usually arising from misplaced remnants of the Wolffian body, Mullerian duct, or possibly pancreatic tissue; (5) serous or serosanguinous cysts resulting from hemorrhage in the mesentery; and (6) enterocystomas which are really not mesenteric cysts since they are outpouchings of the intestinal wall.

The most familiar type of cysts and most common form of primary tumor of the mesentery is the cavernous lymphangioma or chylous cyst. The pathologic picture presented by this lesion is not complex. It may be simple or multilocular, the wall is composed of fibrous and elastic tissue, the cavity lined with flat endothelium and the contents either mucoid, watery, milky, or bloody. The etiologic basis is a pathologic dilatation of the mesenteric lymph vessels re-

sulting from a developmental disturbance of the subepithelial lymph spaces; these cysts usually appear before the age of thirty years.

Mesenteric cysts are generally smaller than an egg, but may be fairly large. When large enough to be palpable the physical signs are rather characteristic. The cyst lies within the circle of the colon; it is firm, well-encapsulated, movable, especially laterally; it may have an overlying tympanitic loop of bowel. The cyst does not move with respiration, does not consistently transmit the pulsations of the aorta, does not extend towards the loin and it does not cause characteristic blood changes.

In the differential diagnosis one must consider enlarged spleen, ovarian cyst with a long pedicle, kidney tumor, pancreatic cyst, retroperitoneal tumor, and perforated diverticulitis with abscess formation.

The ideal treatment is simple excision which is usually possible. Occasionally the resulting trauma will necessitate resection of the bowel. If this appears likely and the surgical risk too great, the cyst may be incised rather than excised and then marsupialized and its cavity packed with gauze. Aspiration has been recommended for the hemorrhagic type, but this seems to be of only questionable value. There appears to be little if any indication for irradiation therapy.

I have seen two patients with primary tumors of the mesentery, each had a cyst and each case was interesting from a different viewpoint.

REPORT OF CASES

CASE 1.—This has previously been reported in part. When Miss E. W. was first seen in consultation with Dr. C. F. C. Hancock, in 1926, she was 47 years of age. Her chief complaint was pain and a lump in the left upper portion of the abdomen. Her previous health had been excellent, no serious illnesses and no operation. Her illness at that time had begun nine months earlier with belching, nausea, vomiting, a weighty feeling in the abdomen and finally a palpable mass in the left upper quadrant. Gradually she had lost her appetite, become constipated and had experienced some shortness of breath. There was constant tenderness over the mass and occasional sharp pains. She had been confined to bed for two weeks preceding her entrance to the hospital.

Physical examination was essentially negative except for an easily palpable mass in the left upper quadrant. It was about 5 inches in diameter, rounded and smooth in contour, encapsulated, movable; it pointed forward in the abdomen rather than laterally towards the loin. It was not especially tender, not notched, and not closely apposed to the rib margin.

Urine examination showed no sugar, albumin, blood, or pus. The red count was 4,000,000 with 80 per cent hemoglobin, the white count 8,250 with 72 per cent polymorphonuclears. The x-ray examination showed a normal duodenal cap, and the stomach contour smooth but its cavity compressed in size at the central portion probably by an extra-gastric tumor.

A preoperative diagnosis of intra-abdominal cyst of undetermined origin was made. Under anesthesia the mass disappeared but the operation was pro-

ceeded with since the tumor had been too evident to be a phantom one. A high left rectus incision was made. The peritoneal cavity was found to contain a fairly large amount of clear, thin, straw-colored fluid. Firmly attached to the mesentery of the transverse colon near the splenic flexure was the ruptured wall of a mesenteric cyst. Since the sac wall was so widely and firmly attached to the mesentery that it seemed that excision would necessitate colonic resection which her general condition did not seem to warrant, it was marsupialized. The patient made an uneventful recovery and the sinus closed promptly. While she was never entirely free of some pain and tenderness at the site of the scar she was able to resume and continue active work until about twenty months after the operation.

Following some dietary indiscretion she had acute colicky pains in the upper abdomen, constant nausea, frequent vomiting and distention. These symptoms persisted for one week before she was readmitted to the hospital.

The essential physical findings were limited to the abdomen. The scar of her previous operation puckered at the lower angle and there was tenderness throughout its length. The entire abdomen was moderately distended. There was generalized tenderness and rigidity across the upper abdomen on the left side. No mass was palpated.

The urine again was negative except for a trace of acetone. The red count was 3,789,000 with 73% hemoglobin. The white count was 11,600 with 77.5 per cent polymorphonuclears. The x-ray examination was reported as follows: "Barium enema showed considerable irregularity of the colon at the splenic flexure and upper part of the descending colon. There was considerable delay but no marked obstruction to the passage of barium through the splenic flexure. Remaining portions of the colon filled promptly. The cecum, descending colon, and rectum were somewhat dilated. A flat plate of the abdomen showed no renal or biliary calculi." A preoperative diagnosis of incomplete obstruction from postoperative adhesions was made.

The scar of the previous operation was excised and a few peritoneal adhesions of no consequence were found. In the mesentery of the transverse colon there was a cyst the size of a small grapefruit. From it there extended to the parietal peritoneum a fibrous band. This cyst was easily enucleated from the mesentery. A tiny tear in the sac released a small amount of thick dark brown fluid. The rent in the mesentery was easily sutured.

The pathologic report was particularly interesting.

"Gross Description. The specimen consisted of a cyst 110 by 70 by 15 mm. Surface pinkish to red. Wall grayish, thickened and friable. Inner lining reddish brown and somewhat necrotic looking.

"Microscopic Description: Two sets of sections showed diffuse fibrous tissue with necrosis, congestion, hemorrhage and marked reaction of leukocytes, chiefly lymphocytes, although endothelials were numerous, mostly filled with brownish granular pigment. There were some scattered polymorphonuclears, also fairly numerous eosinophils in some places. Scattered through the sections were two types of glandular structures. One was lined with a single layer of columnar epithelium and looked like cross-sections of ducts with marked reaction of polymorphonuclears and round cells within and around. Elsewhere were clumps of glands suggesting pancreatic alveoli, but these were atypical and often presented solid masses of small epithelial cells with definite hyperchromatic round to oval nuclei. There were scattered mitoses fairly numerous.

"MICROSCOPIC DIAGNOSIS: (1) Adenocarcinoma (alveolar structure suggests pancreatic origin but this is not definite), (2) Subacute and chronic inflammation."

The wound healed satisfactorily but the patient's general recovery was very slow. In time, however, she resumed her regular activities. Her course, subsequent to this time, has not been previously reported.

On two occasions since, first twenty months and secondly ten years after the second operation, laparotomy was required for incomplete obstruction of the jejunum. At each operation inspection of the scar in the mesentery of the transverse colon showed complete healing and no evidence of recurrence of the
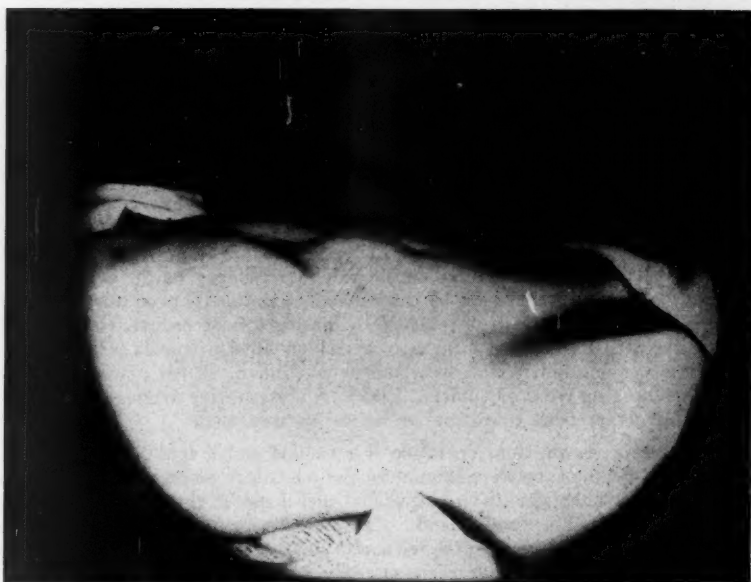
Fig. 1. Pointer shows bulging mass above the umbilicus (Case 2).

cyst. A small mass palpable at one time preoperatively was found to be a dilated loop of jejunem. At the most recent examination some months ago no mass was palpable.

CASE 2.—E. C., referred by Dr. R. I. Kerr, was more satisfactory from all viewpoints, diagnosis, treatment, and progress. He was a white mechanic, 19 years of age, whose chief complaint was a lump in the abdomen. His previous health had been excellent without serious illness or operation. The present illness had begun about a year earlier when he first noticed a lump in the abdomen. It gradually increased in size and remained movable. At times there was epigastric distress relieved by the ingestion of soda water. No real indigestion or severe pain was noted but there was a loss of 8 pounds in two months.

Physical examination was negative except for scattered small lymph nodes and the abdominal mass. This mass, about the size of a small grapefruit, was

quite apparent when the patient lay on his back. It was located principally slightly above and to the left of the umbilicus; it was well encapsulated, rounded, firm, and freely movable especially laterally. It did not move with respiration and there was no extension towards the loin.

The urine was negative except for a faint trace of albumin. The red blood corpuscles were 4,250,000 and hemoglobin 82 per cent, the white blood cells,
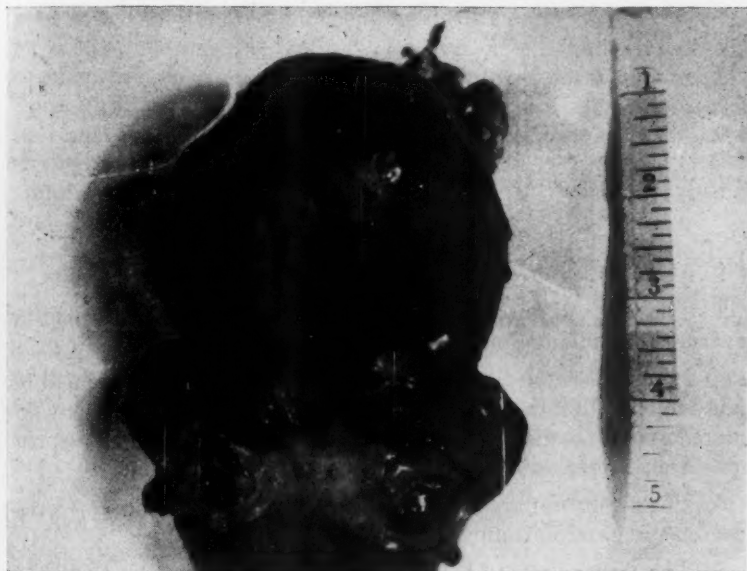
Fig. 2. Cyst removed from mesentery of jejunum.

9,800 with polymorphonuclears 72 per cent. The Kahn test was negative. X-ray examination was reported as follows:

"Stomach normal in size but showed upward displacement and pressure deformity from extrinsic mass. The duodenal bulb showed spastic deformity but could be filled. No six hour residue but transverse colon then showed upward displacement. Marked spasticity of the descending colon at twenty-four hour examination.

"CONCLUSICNS: Abdominal tumor with pressure deformity of the stomach possibly mesenteric or pancreatic cyst."

A preoperative diagnosis of mesenteric cyst was made. A five-inch high left rectus incision was employed. Exploration of the tumor showed it to be a mesenteric cyst between the layers of the mesentery of the jejunum. The tumor was the size of a small grapefruit and was constricted in its central portion. The mesenteric vessels were displaced by the tumor towards one leaf of the mesentery leaving the opposite side rather avascular. The peritoneum of this latter side was incised and the cyst dissected free without rupture. No damage to the mesenteric vessels was apparent and the rent in the mesentery was easily closed with catgut sutures. The spleen was normal. The appendix was not remarkable but it was excised.

The pathologic report described the tumor grossly as "A cyst dissected from the mesentery measures approximately 150 by 110 mm., having a lobulated appearance and consisting of numerous small to large cystic cavities filled with a thick tenacious somewhat mucoid type of material."

The microscopic description stated that "Sections of the tumor showed a thick fibrous wall with lymphocytic infiltration. Such areas as showed the inner surface of the cyst consisted of a loose stroma supporting irregular channels lined by an endothelial type of cell, which had oval to elongated nuclei and occurred mostly in single layers."

PROVISIONAL DIAGNOSIS: Lymphangioma of mesentery.

The postoperative course was entirely uneventful in the hospital except for rather severe gas pains. The patient has now been working at his former occupation for some months and at his last examination was apparently entirely well with no evidence of recurrence.

SUMMARY

1. Most mesenteric tumors are secondary metastatic deposits.

2. The most common primary tumor of the mesentery is the cavernous lymphangioma or chylous cyst.

3. If the mesenteric cyst is large enough to be palpable it will be found to be within the circle of the colon, firm in consistency, encapsulated in outline, movable, but its motility is not affected by respiration; it may have an overlying tympanitic loop of bowel.

4. The symptoms are not characteristic and will be those of pressure on the gastrointestinal tract.

5. The treatment of all mesenteric tumors unless obviously secondary is abdominal exploration and excision with or without colonic resection. If this is not feasible and the tumor is a cystic one marsupialization may be employed.

6. Two illustrative cases are reported.

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SURGERY IN PEPTIC ULCER

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PEPTIC ULCER is one of the most common diseases to which the human race is subject. Bevan[1] cites the work of Carl Hart of Berlin, who believes that peptic ulcer occurs in 10 to 12 per cent of the population. He bases his opinion on a large series of post-mortem examinations. Portis and Jaffe[2] found the incidence of all types of peptic ulcer, in a series of 9,171 consecutive necropsies, to be 5 per cent. Cleveland and Walters[3] report that in 1938 and 1939 at the Mayo Clinic the incidence of gastric ulcer was 0.24 per cent, and during the same period 3.13 per cent of all patients registered had duodenal ulcer.

While the vast majority of cases of peptic ulcer can be treated satisfactorily by medical means, there are a few in which surgery is demanded, and many in which some form of surgery is the treatment of choice. Marshall[4] states that at the Lahey Clinic 8 per cent of the patients with duodenal ulcer and 23 per cent with gastric ulcer have been subjected to surgery. During the years 1938 and 1939 at the Mayo Clinic, as reported by Cleveland and Walters[3], 16.1 per cent of the patients with duodenal ulcers and 50.1 per cent of the patients with gastric ulcers were treated surgically. In our clinic 25 per cent of the patients with peptic ulcers have had surgical treatment. The percentage of cases treated surgically in the various clinics is much higher than that occurring in the work of the general practitioner, because the peptic ulcer cases treated by the former have frequently been referred to them because they have failed to respond to medical treatment.

Internists as well as surgeons agree that many cases of peptic ulcer should be treated surgically. We have all come to realize that the surgeon and the internist should cooperate instead of compete in their therapeutic efforts.

Until comparatively recently, in this country, gastroenterostomy and pyloroplasty were the operations most commonly used in the surgical treatment of peptic ulcers, especially those effecting the duodenum; the most radical operation, partial gastrectomy, being reserved for certain gastric ulcers. Lately there has been a trend toward more radical surgery in all types of peptic ulcer so that now partial gastrectomy is frequently the operation of choice for duodenal as well as gastric ulcer.

INDICATIONS FOR SURGERY

The indications for surgery in peptic ulcer, as generally accepted, are as follows:

1, perforation,

2, certain cases of hemorrhage,

3, cicatricial and recurring types of pyloric obstruction,

4, intractable duodenal ulcers,

5, intractable and questionable malignant gastric ulcers,

6, gastrojejunal ulcers,

7, and cases in which patients will not or cannot take proper medical treatment on account of economic or other reasons.

While the indications as given above are well known, and usually accepted, personal opinion, experience and judgment must guide us in deciding just when medical treatment will be discontinued and surgery instituted. It is our practice to advise surgery in all cases that have not responded to adequate medical treatment in a reasonable length of time. We are opposed to allowing the patient to continue over a relatively prolonged period in an invalid or semi-invalid state ever in danger of perforation, hemorrhage, pyloric obstruction and malignant changes.

PERFORATION

Perforation of a peptic ulcer *into the general abdominal cavity* may develop very suddenly or it may develop slowly and be sealed by some abdominal structure. Hunt[5] speaks of the latter as a "protective perforation," and states that it occurred in 13.8 per cent of the duodenal and 32 per cent of the gastric ulcer cases in his series requiring surgery. While these latter cases do not require emergency surgery they are very resistant to medical treatment and are frequently cured only by some surgical procedure.

Acute perforation is one of the most urgent of the surgical emergencies: it will usually result in a fatality if it is not recognized early and appropriate surgical measures instituted. It has been found that if these cases come to surgery within the first six or eight hours after perforation the mortality is comparatively low and that the passage of each hour after this adds progressively to the mortality rate. We agree, however, with Graham[6] that sufficient time should be taken, preoperatively, to treat the patient adequately for shock if it exist.

While suture of the perforation and omental reenforcement is the operation most commonly used for acute perforation of a peptic

ulcer, there are some cases in which it may be better judgment to alter this procedure; and there are some surgeons who do not, as a rule, consider this operation adequate. We believe that the majority of American surgeons agree with Graham[6] when he states, "The fundamental surgical principle applicable to all emergency surgery— that the patient be treated solely for the lesion creating the emergency—should be observed to a greater degree in the treatment of patients suffering from an acute perforation of a duodenal ulcer than in almost any acute intraperitoneal lesion." Since the vast majority of the peptic ulcers which perforate are on the anterior wall of the duodenum, and many patients do well on medical treatment after simple closure of the perforation most surgeons think that any radical operation at the time of the perforation is meddlesome surgery.

Cases are occasionally encountered in which the perforation is such that closure by sutures is impossible. Under these circumstances satisfactory results may be obtained by simply suturing omentum over the perforation. Graham uses this method routinely. He makes no attempt to suture the perforation itself, but fixes a piece of omentum over it by interrupted sutures. Finsterer[7] advises, in certain of these cases, that the end of a small rubber tube be inserted into the perforation and the other end be allowed to escape from the incision. He then wraps omentum around the tube from the perforation to the abdominal wall. After a few days the tube is removed and he states that the opening rapidly closes.

Since the majority of perforated peptic ulcers are on the anterior wall of the duodenum or stomach near the pylorus, it is frequently easy to excise the ulcer and do some type of pyloroplasty. Bevan[1] strongly recommends this procedure. He removes the ulcer by a diamond-shaped incision and in so doing also removes the anterior half of the pyloric muscle. The pyloroplasty is then carried out very much like the original Heineke-Mukulicz operation.

In two cases of perforated duodenal ulcer I have excised the ulcer and combined with it pyloroplasty according to the technic of Horsley. Excellent results were obtained in these cases. I believe that this operation is rarely indicated and have not used it now for several years.

Closure of the perforation combined with gastroenterostomy has been extensively used in the past. This procedure was strongly advised by the late Dr. Deaver of Philadelphia. In his hands the results were apparently satisfactory, but in average hands, I believe that it should be used only when the existing conditions demand it.

I have performed gastroenterostomy on very few occasions at the time of closure of the perforation, using it only when I felt it was indicated on account of marked pyloric obstruction. In several instances although there was rather marked pyloric obstruction after closure of the perforation I decided to take the risk and not do a gastroenterostomy. In none of these cases have I regretted the decision. I believe that marked pyloric obstruction is the only indication for gastroenterostomy at the time of closure of the perforation.

Many European surgeons resort to partial gastrectomy as a routine in the treatment of acute perforation of peptic ulcer if the condition of the patient will permit. Judin[8], of Moscow, reports 331 partial gastrectomies for perforated ulcer with a mortality of only 7.8 per cent. This, of course, is an enviable record. I agree with the majority of American surgeons that this operation is indicated only under rather rare circumstances. On only one occasion have I resorted to gastric resection in perforated ulcer. Good results were obtained. Pathologic study proved this to be a carcinomatous lesion.

HEMORRHAGE

Statistics indicate that gross hemorrhage from peptic ulcer is a rather common occurrence. The majority of observers feel that perforation occurs more frequently than hemorrhage, but in Goldman's[9] series of cases hemorrhage occurred in 39 per cent and perforation in 23 per cent. Pfeiffer[10] found the incidence of bleeding in his series of peptic ulcers to be 18 per cent, and Lahey[11] reports that 18 per cent of the duodenal ulcers and 11 per cent of the gastric ulcers in his series had gross hemorrhage. Hinton[12] found that in a large series of peptic ulcers under medical treatment only 3.3 per cent developed gross hemorrhage.

While many physicians believe that the mortality due to hemorrhage from peptic ulcer is very low, statistics show this to be a falacy. Of Goldman's patients 11.1 per cent died and Pfeiffer reports a mortality rate of 12.9 per cent. Marshall and Keifer[13] give the death rate at the Lahey Clinic at 4.6 per cent. J. M. Blackford and Williams[14] reviewed all the deaths recorded with the Seattle Bureau of Vital Statistics over a five-year period and found that there were 116 deaths due to hemorrhage from peptic ulcer among the 23,955 deaths recorded. It was also noted that 97 per cent of the patients who died of hemorrhage from peptic ulcer were over 45 years of age. All writers who discuss this phase of the subject call attention to the fact that the mortality rate in patients over 40 years of age is much higher than in those in the younger age group.

It has been our experience that the majority of patients who have had one or more gross hemorrhages do not respond well to medical management. Lahey[15] reports that Kiefer, of his clinic, has demonstrated that about 40 per cent of the patients who had one hemorrhage failed to respond satisfactorily to medical treatment; and that 80 per cent of those having two or more hemorrhages required some type of surgery for relief of symptoms.

Finsterer[16] advised the routine practice of surgical treatment in severe or massive hemorrhage, and urged that it be carried out within the first 24 or 48 hours—before irreparable damage has taken place in the vital organs as a result of the anemia. The majority of surgeons agree that there is the occasional case in which early surgery is necessary if the life of the patient is to be saved. It has been shown that the mortality is much less in these cases if surgery is carried out within the first 48 hours, provided the symptoms cannot be much improved by repeated blood transfusions and other non-operative treatment. I believe that in the majority of cases bleeding can be controlled and the condition of the patient much improved before resorting to surgery.

Occasionally bleeding from peptic ulcer takes place while the patient is taking proper medical treatment. I believe that, in these cases, as soon as the general condition of the patient will permit, surgery should be advised. I do not subscribe to the teaching that we should wait until the patient has two or more hemorrhages before advising surgery, if the bleeding occurs while the patient is under adequate medical supervision. Those patients who have had repeated hemorrhages and have not been under adequate medical supervision should be advised to have operation if they are unable or unwilling to take proper medical treatment.

Various surgical procedures have been used in the treatment of bleeding ulcer. While it is generally agreed that the bleeding ulcer itself should be attacked, it is possible that occasionally this is not practicable. In these cases one of the indirect operations, such as gastroenterostomy or pyloroplasty, may be done with good results, but these are certainly not operations of choice. If a bleeding ulcer is on the anterior wall near the pylorus, excision of the ulcer with pyloroplasty would be very appropriate. This, however, is a rare occurrence. A bleeding ulcer of the stomach on the lesser curvature can frequently be excised and gastroenterostomy carried out. This is a satisfactory procedure if there is no evidence of malignancy. Occasionally it is possible to ligate the bleeding vessels and combine with it pyloroplasty or gastroenterostomy. I think, however, that the ulcer itself should always be removed if possible.

It is my opinion that the operation of choice in bleeding ulcer is partial gastrectomy. In the majority of cases the ulcer which bleeds is either on the posterior wall of the duodenum or along the lesser curvature of the stomach. On account of the inaccessibility of ulcers on the posterior duodenal wall, and the danger of carcinoma in gastric ulcer partial gastrectomy offers the best opportunity for permanent and complete recovery.

PYLORIC OBSTRUCTION

Some type of pyloric obstruction is probably the most common complication of peptic ulcer. Judging from the retention of barium sulphate or food in the stomach beyond the normal time, Kruse[17] found that 51 per cent of his ulcer cases showed some obstructive characteristics. On further analysis, however, it was shown that only 20 per cent had cicatricial organic changes. While these cases usually do not present the dramatic features frequently observed in some of the other complications of peptic ulcer, they may become serious and carry a moderately high mortality rate. In their post-mortem studies Portis and Jaffe[2] found that death was due to pyloric obstruction in 11.6 per cent of deaths as a result of peptic ulcer.

Since it is frequently impossible to know at first whether the symptoms are due to muscle spasm, acute inflammatory changes or cicatricial contracture, it is important that these patients be kept under observation for a time to note the effect of medical treatment. I believe that surgery is indicated in comparatively few of the cases presenting symptoms of pyloric obstruction. Practically all of the cases due to pylorospasm and acute inflammatory changes and a small number of these having cicatricial narrowing can be relieved or greatly improved by medical treatment. Surgery should be reserved for those patients having irreversible changes who cannot live in comfort on a reasonably liberal diet, and also the occasional patient who has recurrent attacks of obstruction due to pylorospasm or acute inflammatory changes even with good medical care.

The type of surgery indicated in the individual case depends upon the pathology and pathologic physiology which exists. Pyloroplasty, gastroenterostomy and partial gastrectomy are the operations most commonly used for this complication of peptic ulcer, and each has its definite indications.

Some type of pyloroplasty may give satisfactory results in the occasional case of pyloric stenosis. I think that it should be used only in those cases with a low gastric acidity. Since in the majority of these cases there is a large amount of scarring this operation is usually not satisfactory.

Partial gastrectomy is now being used in this type of obstruction more than formerly. I believe that this operation gives more satisfactory results than does gastroenterostomy when the obstruction is associated with an active duodenal ulcer with a high gastric acidity. If obstruction is due to an active ulcer partial gastrectomy is advisable on account of the danger of malignancy. Occasionally pyloric obstruction is associated with a bleeding ulcer. Since the ulcer should be removed in these cases, here too, I consider partial gastrectomy the operation of choice.

On the other hand where pyloric obstruction is due to duodenal ulcer in a comparatively old individual, and the ulcer has healed or become rather inactive and the gastric acidity is low, gastroenterostomy can be used with excellent results.

INTRACTABLE DUODENAL ULCERS

There are many cases of duodenal ulcer without any of the previously mentioned complications which fail to respond properly to medical treatment. These patients may pass into a state of chronic invalidism and are constantly in danger of perforation, hemorrhage, and pyloric obstruction. Just when surgery should be advised is a question requiring expert judgment. There are cases which show little improvement at any time under medical treatment, and others which do well for a time, but have frequent exacerbations of activity. No patient should be considered successfully handled medically until he shows no evidence of ulcer as demonstrated by symptoms, laboratory and x-ray findings.

There are many factors which determine the operative procedure to be carried out in an individual case. Age, sex, race, occupation, the emotional stability of the individual and his general physical condition are due consideration. It is well to reserve final decision as to the operation to be performed until the abdomen is opened and one has had an opportunity to determine the exact pathologic condition and the ease with which one of the various procedures may be carried out. It is only by such consideration that we may obtain the best results.

Pyloroplasty has been extensively used in the treatment of peptic ulcer: it is considered the safest of the surgical procedures. In this operation the pyloric muscle is sectioned or partly removed and the pyloric opening is widened so that pylorospasm is eliminated, and the emptying of the stomach is facilitated. Occasionally the ulcer, if located on the anterior wall of the duodenum, can be removed as the incision for the pyloroplasty is made. It has the great advantage of allowing the gastric contents to continue to go into the duodenum,

the part of the intestine best suited to protect itself against highly acid material.

Pyloroplasty may be technically difficult if the duodenum is immovable and badly scarred. The acidity of the stomach is usually little changed by this operation, so those cases having a high gastric acidity are better treated by some other procedure. I think this is also true of the penetrating type of ulcer and many of those on the posterior wall of the duodenum.

Gastroenterostomy is the best known of all stomach operations, and is usually not difficult to perform. It can be done on poor risk cases with a comparatively low mortality rate. Priestly[18] reports a series of cases in which this operation was carried.out for duodenal ulcer with a mortality rate of 0.8 per cent, and a larger series with a mortality rate of 2.1 per cent.

The physiology is considerably changed after gastroenterostomy. The material from the stomach finds its way into the intestines both through the gastroenterostomy stoma and the pyloric opening (in the absence of complete pyloric stenosis). Gastric acidity is reduced to a variable degree depending upon the influx of intestinal juices into the stomach.

There are two definite disadvantages to this operation: (1) the ulcer is usually not removed when the operation is done for duodenal ulcer, and may continue to give trouble, and (2) jejunal or gastro-jejunal ulcer frequently develops. Walters and Clagett[19] state that gastrojejunal ulcer occurs in 3.2 per cent of the cases following gastroenterostomy at the Mayo Clinic. Hinton[20] reports the incidence of this complication to be 16.4 per cent in his series. Lahey[21] believes that a reasonable average estimate of the incidence of gastrojejunal ulcer following gastroenterostomy for peptic ulcer is about 15 per cent. On account of these disadvantages, I believe that this operation should be reserved for the poor risk cases and those in the older age group with a low gastric acidity.

In recent years partial gastrectomy has gained rapidly in popularity in the treatment of these cases, and I believe deservedly so. Until quite recently one-half or less of the stomach was removed, but now the tendency is to be still more radical and most surgeons advise the removal of two-thirds to four-fifths of the stomach.

The physiology is changed more by this than any of the other operations for peptic ulcer. In this operation, as now commonly done, the pylorus along with a large part of the acid-producing portion of the stomach is removed, usually, also, with the portion of the duodenum bearing the ulcer. The gastric acidity is markedly

lowered or an anacidity may exist due both to a removal of a considerable part of the acid-producing portion of the stomach, and the neutralizing effect of the intestinal juices which enter the stomach through the gastrojejunal stoma. It is considered that this lowered gastric acidity or anacidity is largely responsible for the low incidence of gastrojejunal ulcer and the excellent results obtained by this operation.

Unfortunately, in spite of the more radical gastric resections, gastrojejunal ulcers still occur. However, the incidence is much lower than that following gastroenterostomy. Lahey[21] states that gastrojejunal ulcer occurs in about 2.25 per cent of his partial gastrectomies and Walters, Lewis, and Lemon[22] report a series in which it occurred in 3 per cent.

A high mortality rate is the main basis for criticism of this operation for duodenal ulcer. While the mortality rate is usually considered to be from 5 to 10 per cent, lower figures have been presented by several surgeons. Graham[23] reports a mortality rate of 3.8 per cent, and Walters, Lewis and Lemon[22] report a rate of 1.9 per cent in a series of 212 cases. It is generally considered that the safety of the operation is enhanced if the resection be carried out proximal to the pyloric ring. This is especially true where the ulcer penetrates deeply into the pancreas and is associated with marked inflammatory changes. I believe that the mortality rate in this operation can be kept within reasonable limits by a judicious selection of cases, painstaking preoperative preparation and careful technic at operation.

It is my opinion that partial gastrectomy is the operation of choice for all cases of duodenal ulcer without demonstrable complications requiring surgery if the condition of the patient will permit, and if the lesions and anatomic variaitons founds on exposure are such that the operation can be done with comparative ease.

INTRACTABLE AND QUESTIONABLY MALIGNANT GASTRIC ULCER

Since many gastric ulcers are found to be carcinomatous on pathologic study, our responsibility is much increased when treating these cases as compared to duodenal ulcer. Cleveland and Walters[3] report that 19 per cent of their presumably benign gastric lesions were malignant. It is generally held that every ulcer of the stomach should be considered a potentially malignant lesion.

Many gastric ulcers will respond well to medical treatment. In favorable cases the pain is soon relieved, occult blood rapidly disappears from the stool and the ulcer niche, as seen on x-ray exami-

nation, diminishes in size in a short time. Scott and Mider[24] state that the niche should decrease at least one-third of its cross-section area within three weeks. Patients who respond in this manner may be continued on a medical regimen. However, they should be kept under close observation, and if the ulcer does not entirely disappear operation should be advised. Since occasionally the ulcer recurs at the site of the original lesion after all evidence, including symptoms, laboratory and roentgenologic study, has disappeared, these cases should be kept under observation over a long period of time.

Any case which does not respond promptly to medical treatment is placed in the intractable or carcinoma-suspect group, and surgical treatment is advised.

Since any gastric ulcer which does not heal promptly on medical regimen may be carcinomatous, I believe that partial gastrectomy is the most appropriate operation. The more conservative operations, such as gastroenterostomy and excision of the ulcer combined with gastroenterostomy, should be reserved for only those cases whose general condition or some local condition renders gastric resection too hazardous.

Gastrojejunal Ulcer

Gastrojejunal ulceration is usually a complication of the surgical treatment of peptic ulcer, most commonly duodenal. However, it may develop after any anastomosis of the stomach to the jejunum. On account of the seriousness of this condition and its complications it has occasioned much study and considerable controversy.

In the past it was suggested that various factors in the technic of gastrojejunal anastomosis, such as the use of non-absorbable suture material and trauma due to pressure from clamps, were responsible for the development of these ulcers. It is now generally agreed that these factors are of little importance. At the present time it is the opinion of the majority that tissue susceptibility to ulcer formation and gastric acidity are the most important causes. It has been repeatedly shown that young, highstrung, nervous individuals with high gastric acidity are particularly prone to develop this complication, and that certain races are especially susceptible.

Statistics show that gastrojejunal ulcer occurs considerably more often following gastroenterostomy, as the treatment for duodenal ulcer, than it does following partial gastrectomy; and the more radical the gastric resection the less likely is the occurrence of this complication. This condition rarely follows the surgical treatment of gastric ulcer.

In addition to the pain and digestive symptoms which are produced by this condition, hemorrhage, perforation and gastrojejunocolic fistula frequently occur. Walters and Clagett[19] report that gastrojejunocolic fistula occur in about 14 per cent of the gastrojejunal ulcer cases seen at the Mayo Clinic. These patients with gastrojejunocolic fistula rapidly become debilitated on account of a large amount of food passing directly into the colon without an opportunity for absorption in the small intestine. This adds greatly to the risk of the formidable surgical procedure which is necessary for its cure.

Gastrojejunal ulcer is usually intractable to medical management. Walters and Clagett state that surgical treatment should be advised when the diagnosis is made. Lahey[21] believes that these patients should be submitted to a trial at medical treatment. It is generally agreed that if very prompt relief is not obtained by non-operative attempts, surgery should be advised so that the more serious complications may be prevented. The importance of early surgical treatment is realized when we compare the mortality rate of the uncomplicated with that of the complicated cases. Finsterer[26] reports an operative mortality rate of 12.2 per cent for all uncomplicated gastrojejunal ulcers and in the complicated cases the mortality rate was 36.8 per cent for gastrojejunocolic fistula and 36.3 per cent for profuse hemorrhage.

Since patients with gastrojejunal ulcer have a predisposition to ulcer formation and usually a high gastric acidity, in spite of the original operation, most surgeons are of the opinion that the gastric physiology must be definitely and permanently changed if a cure is to be attained. At the present time partial gastrectomy is the operation of choice by the majority of surgeons if the gastrojejunal ulceration follows gastroenterostomy and a more extensive gastric resection if partial gastrectomy was the original operation. Crile[27] believes that many of these cases can be cured by changing the temperament of these individuals "to a lower tempo," by adrenal-sympathetic surgery.

SUMMARY AND CONCLUSIONS

1. Statistics indicate that peptic ulcer occurs in about 5 per cent of the population.

2. The indications for surgery are as follows: Perforation, certain cases of hemorrhage, cicatricial and recurring types of pyloric obstruction, intractable duodenal ulcer, intractable and questionably malignant gastric ulcers, cases which cannot or will not take proper

medical treatment on account of economic or other reasons, and gastrojejunal ulcer.

3. Early surgery is essential in acute perforation of peptic ulcer. Simple closure of the perforation is usually the operation of choice.

4. A large per cent of patients having gross hemorrhage from peptic ulcer will respond to medical treatment. In the vast majority of the cases in which surgery is indicated the bleeding can be controlled and the condition of the patient much improved before surgery is resorted to. The indirect operations, such as pyloroplasty and gastroenterostomy are rarely indicated in bleeding peptic ulcers. Partial gastrectomy is the operation of choice if the condition of the patient will permit.

5. Some type of pyloric obstruction is probably the most common complication of peptic ulcer. A comparatively small per cent, however, is of the type or degree that requires surgical treatment. In a large per cent of the cases requiring surgery gastroenterostomy is the operation of choice. Partial gastrectomy is preferable in those cases having a high gastric acidity and those having an active gastric ulcer.

6. A fairly large per cent of duodenal ulcers, without demonstrable complications, fail to respond to medical treatment, and therefore surgery is indicated. Pyloroplasty, gastroenterostomy and partial gastrectomy are the operations commonly used. Since the physiology of the stomach is not sufficiently altered by pyloroplasty and gastroenterostomy, the operation of choice in the majority of cases associated with high gastric acidity is gastric resection.

7. Gastric ulcers which fail to respond readily to medical treatment should be subjected to surgery. Due to the large per cent of gastric ulcers found to be carcinomatous on pathologic study, partial gastrectomy is indicated in these cases. Gastroenterostomy and excision of the ulcer with gastroenterostomy should be reserved for those patients whose general condition is poor or who have some local condition which would render the more radical operation too hazardous.

8. Gastrojejunal ulceration is usually a complication of the surgical treatment of peptic ulcer, most commonly duodenal. Tissue susceptibility to ulcer formation and high gastric acidity are the most common causes. It occurs most commonly following gastroenterostomy for duodenal ulcer and less frequently follows gastric resection. The more radical the resection the less likely the devel-

opment of gastrojejunal ulceration. Partial gastrectomy should be done if the ulcer follows gastroenterostomy and a more extensive gastric resection if partial gastrectomy was the original operation.

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TRAUMATIC INTRACEREBRAL PNEUMATOCELE

Report of a Case

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AIR occasionally enters the cranial cavity by way of a fracture which extends through the nasal sinuses or cribriform plate. The air may be forced in by coughing, sneezing or blowing of the nose. Also air may be sucked into the cranium during periods of low intracranial pressure in patients with profuse cerebrospinal rhinorrhea. The air finds its way into the subdural space, the subarachnoid space, the ventricles or the brain tissue. The presence of intracranial air has been variously designated as pneumocephalus, pneumoencephalon, pneumatocele, pneumoaerocele, pneumocranium, cerebral emphysema and pneumoventricle. Chiari[1] was the first to report a case in 1884. In 1926, Dandy[2] could only find 25 cases in the literature. Since then, cases have been diagnosed more often and by 1935, Plunkett and Lendrum[3] collected 101 cases from the literature. That the condition occurs even more frequently is suggested by the fact that Dandy[4] has seen 15 cases. Intracranial air may cause mild or severe symptoms. Headache can be the only complaint. Later signs and symptoms suggesting brain tumor may appear. The diagnosis can be established by roentgen films. Dandy[4] estimated that if untreated 40 per cent of these patients die. In Plunkett and Lendrum's collected series including both treated and untreated cases, the mortality rate was 30 per cent. Death results from increased intracranial pressure, meningitis or brain abscess. It is not desirable to await spontaneous cure (which occurred in 28 per cent of the reported cases according to van Zyl[5]) because relief of symptoms can be obtained surgically with less risk. Treatment consists of craniotomy and obliteration of the fistula.

REPORT OF A CASE

J. F., a white male, aged 26, was brought to Charity Hospital in New Orleans on June 20, 1940, because of convulsive seizures and increasing weakness of the left upper and lower extremities. A presumptive diagnosis of brain tumor was made.

On March 3, 1937, while crossing a street, the patient was struck down by an automobile, receiving a blow on the forehead which rendered him unconscious for approximately ten minutes. He was taken to a hospital where lacerations were sutured. Films of the skull revealed a fracture of the frontal bone. He remained under observation for one week during which time he

Fig. 1—Lateral film showing pneumatocele within the frontal lobe

complained only of frontal headache of increasing severity. If there was a cere-brospinal rhinorrhea, the patient did not notice it. After dismissal from the hospital, the patient returned to his job as an iron worker despite the headache. No additional complaints appeared until sixteen and one-half months after the accident, at which time the patient experienced two mild convulsive seiz-ures unassociated with loss of consciousness. A third seizure on the same day rendered the patient unconscious. Two months later the patient noted a "pat-ting" of the left foot when he walked and a weakness of the left upper ex-tremity. The headache and the weakness of the left side of the body persisted until May, 1940, when, following a severe convulsive seizure, the first in seven months, the patient came to Charity Hospital.

Physical examination showed several small scars over the forehead and slight atrophy of the muscles of the left upper and lower limbs. Neurologic examination revealed little more than a weakness of the muscles of the left side of the lower half of the face as well as of the left arm and leg. The ten-don reflexes on the left side were hyperactive. There was no choking of the optic disks. Roentgenograms of the skull disclosed a large air-filled cavity in the right frontal lobe of the brain.

A diagnosis of traumatic pneumatocele having been established, craniotomy was performed on July 10, 1940. An osteoplastic flap was reflected under local anesthesia, exposing a cystic frontal lobe. On opening the cyst and in-serting a lighted retractor it was possible to inspect the entire lining of the cavity. It was smooth-walled and presented a single deep dimple in the in-ferior, anteromedial region which was obviously the opening of a sinus. The

Fig. 2—Postero-anterior film showing the pneumatocele

cyst contained no fluid. The frontal lobe was next elevated from the floor of the anterior fossa exposing a string of gelatinous tissue 2 cm. in length lying beside the nerve in the olfactory groove and connecting the under surface of the frontal lobe with the cribriform plate. The fistulous tract was coagulated electrically and excised. The minute opening in the dura and cribriform plate was coagulated and a piece of temporal muscle was packed against this region. After permitting the frontal lobe to fall back into place, the dura was closed, leaving the collapsed cyst and subdural space filled with saline. The bone flap was replaced and the scalp tightly closed.

Except for several convulsive seizures during the first three and one-half weeks following operation, the patient's course was uneventful though slow and he was discharged six weeks later. He stated that his left limbs were stronger than on admission. Nine months following the operation, the patient was working as an oil driller and was perfectly well.

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IMPORTANCE OF VITAMIN THERAPY IN THE PREPARATION AND POSTOPERATIVE CARE OF SURGICAL PATIENTS

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Augusta

MALNUTRITION in the sense of avitaminosis is quite as much a problem for surgeons as for practitioners of internal medicine. Very many patients who require surgical treatment come from the large portion of the population which from necessity subsists on an inadequate or borderline diet. Many others who are well able to procure proper food present themselves in an advanced stage of depletion because they have long had bad food habits or have followed some dietary fad. Some have remained for months or years on severely restricted diets prescribed for the relief of allergy or of functional or organic disease of the gastrointestinal or biliary tract. In addition to these, there are the people with anorexia often of nervous origin, sometimes due to organic disturbances; and the alcoholics who are chronically on the borderline of deficiency disease. Such defects in food intake are usually obvious. Equally important is failure of absorption of essential dietary components when an adequate diet is taken. This may result from inadequate digestion in patients with achlorhydria or from hurried transit through the upper intestine in hyperperistalsis with diarrhea or from lack of bile in hepatic or biliary disease. Frequent vomiting from any cause is another common source of failure of absorption of vitamins which is often overlooked. The liver is the storehouse of all the vitamins which are known to be important in human nutrition so that failure of utilization and storage is apt to occur in any type of hepatic disease regardless of the adequacy of the diet. Increased metabolic demand for vitamins is present in every case of hyperthyroidism, whenever there is fever and always when patients are maintained on parenteral injection of glucose solutions. In patients depleted from any cause operative procedures may precipitate frank symptoms and signs of deficiency disease.

The general patterns of avitaminoses which may exist are determined to a considerable extent by the factors just mentioned. In a very broad sense the prolonged use of a diet excessively high in carbohydrate, the loss of food from vomiting or diarrhea and the presence of hypermetabolism result in clinical manifestations of deficiency of the water-soluble vitamins; hepatic and biliary diseases

are apt to be associated with evidences of lack of the oil soluble group. The manifestations of nutritional disease presented by surgical patients are apt to be severe and to develop with great rapidity. We are always likely to consider them an acute complication of the preoperative or postoperative period and to try to relate them to a single nutritional factor. It is much wiser and probably much more accurate to regard such episodes as evidence of extreme depletion, developing rapidly it is true, but in individuals with a deep background of chronic nutritional inadequacy. It is probably entirely true that the avitaminoses are never solitary. We are in the habit of attributing certain symptoms and signs to deprivation of a single vitamin, we speak glibly of the relation of thiamine deficiency to peripheral neuritis and to beri-beri, or pellagra being due to nicotinic acid deficiency and of scurvy following inadequate intake of ascorbic acid. While it seems almost certain that the outstanding clinical features of these syndromes depend largely on predominant lack of a single vitamin it is much more certain that each of the major nutritional diseases is a polyavitaminosis.

In spite of the tremendous amount of effort which has been devoted to the study of vitamins relatively little is known of their actual function in the human body or of functional and anatomic changes which can be attributed to specific deficiencies. Much of what we think is true may have to be revised. Vitamin A probably has little direct bearing on surgical problems. There seems to be adequate evidence that the primary functions of this vitamin are to maintain the integrity of specialized epithelium and to regenerate visual purple and visual violet[1,2]. Reputed influence on resistance to infection, wound healing and the formation of kidney stones has not been proved[3,4]. Recognition of signs of deficiency of vitamin A may be of importance in estimating the gravity of biliary and hepatic disease. An adequate supply of bile is required for the absorption of carotene and reasonably good hepatic function is required for the splitting of carotene into vitamin A and proper storage of the vitamin.

The better known vitamins of the B group, thiamine, riboflavin and nicotinic acid are of great surgical interest because of the common incidence of the deficiency states attributed to their lack. These substances are reactive portions of many of the enzymes which are essential in the progressive dehydrogenation of carbohydrates, which is to say the derivation of energy from this important food. The vitamins are gradually used up in the process and unless constantly replaced a grave disturbance of carbohydrate metabolism results. The intake of a great excess of carbohydrate in relation to these vitamins produces rapid depletion with resulting symptoms and

signs which are thought to be specific for deficiency of each substance. The structure of pyridoxin (vitamin B_6) and pantothenic acid makes it likely that they have functions similar to other members of the B group, but as yet no definite place in human physiology can be assigned to them. The liver seems to be the site for storage of all B vitamins and, while it is probable that no considerable reserve is held, hepatic disease is a potent source of deficiency from lack of storage.

Ascorbic acid (vitamin C) is also of much importance in surgery because deficiency results in the tendency to hemorrhage and probably interferes with wound healing. It has long been known that this vitamin controls the formation of intercellular cement substances[5, 6] and the effects of deficiency have been attributed to failure of this function[7, 8, 9]. Vitamin C is also thought to exert a specific effect on erythropoiesis quite different from that of other substances[10], and to have certain enzymatic functions in various oxidation-reduction processes in the body.

Vitamin D has no definite deficiency syndrome in the adult. The chief function of vitamin D seems to be fixation of phosphatase in the bones to secure the presence of adequate amounts of inorganic phosphate for the precipitation of calcium orthophosphate. Deficiency of the vitamin should be suspected, however, in all patients with osteomalacia who have high serum phosphatase values[11]. The status of vitamin E (alpha tocopherol) is still uncertain and no clinical syndrome can be associated with deficiency.

Vitamin K may well be called the surgical vitamin since it has been shown that it is intimately concerned with the production of prothrombin and so with the normal coagulation of blood[12]. Liver damage and biliary obstruction are much more common causes of lack of this vitamin than dietary insufficiency since production of prothrombin is probably confined to the liver cells and absorption of vitamin K is dependent on the presence of bile in the duodenum.

From what has gone before it must be evident that certain groups of patients should be suspected of nutritional deficiency regardless of their apparent preoperative status. On this basis the surgeons of our clinic have become free users of vitamins in the preparation and postoperative care of their patients and the number of consultations requested for deficiency disease has declined notably. Unfortunately the clinical estimation of the patient and therapeutic trial of the vitamins remain the most important tests for avitaminosis. Reasonably satisfactory methods are available for determination of several of the vitamins in the blood and urine but in the case of a majority of these substances there is little variation from normal levels until extreme deficiency is present. All patients from the lower in-

come groups are likely to have latent vitamin deficiency from the use of diets excessively high in carbohydrate. The numerous individuals with peptic ulcer, diverticulitis, regional ileitis and malignant disease of any portion of the gastrointestinal tract may be depleted because of dietary limitations, gastric achlorhydria, vomiting or diarrhea. Those with almost any type of biliary or hepatic disease may suffer from the combined effects of anorexia, vomiting, achlorhydria, diarrhea, inadequate biliary secretion and liver damage. Elderly men with prostatic obstruction are particularly prone to develop avitaminosis from the anorexia and vomiting which so frequently accompany "back-pressure uremia." Patients with lymphopathia venereum involving the colon seem to constitute a special group particularly apt to develop a most severe and intractable form of vitamin deficiency which requires excessive therapeutic dosage. Persons with severe hyperthyroidism, continued fever and those from necessity maintained for considerable periods by intravenous infusions of solutions of glucose constitute a separate group of patients who are apt to become acutely and severely ill as a result of rapid exhaustion of vitamins.

The symptoms and signs of the various avitaminoses are fairly well identified though radical revision of present standards may be required at any time. Vitamin A deficiency in the adult is usually first detected by recognition of the specificity of night blindness and follicular keratosis as signs. The determination of delayed dark adaptation requires the use of some type of adaptometer. Follicular keratosis which is limited to the extensor surfaces of the limbs is characteristic and easily recognized. Determinations of the concentration of carotenoids and of vitamin A in the blood are fairly accurate and not too complicated for use as confirmatory evidence.

Deficiency of thiamine (B_1) is typically characterized by the syndrome of beri-beri. Much more frequently anorexia, vague nerve and muscle pains, slight edema and disturbances of gastrointestinal motility are evidences of the disease. Different investigators using slightly different methods have found that anorexia, vague "neurotic" complaints, constipation and flatulence, tachycardia and edema are the more frequent symptoms and signs produced under experimental conditions[13, 14]. Anorexia, undue fatigue, palpitation and constipation are apt to be complaints of patients with moderate grades of thiamine deficiency. Examination may show increased or diminished reflexes depending on the duration of deficiency, tender nerve trunks, slight loss of tactile and vibratory sensation, slight to moderate enlargement of the heart with poor heart sounds, tachycardia and low arterial blood pressure. X-ray examination shows evidence of marked loss of tone of the gastrointestinal tract which

is corrected by vitamin treatment. Thiamine deficiency is not uncommon as a presenting syndrome in the postoperative period when large amounts of glucose solutions have been given parenterally for hydration and nourishment.

Riboflavin deficiency is not an evident complication in surgical conditions though signs of deprivation may be valuable as indicators of other B group avitaminosis.

Nicotinic acid deficiency seems to be the most frequent nutritional complication of surgical conditions. This is not surprising since the symptoms and signs of lack of nicotinic acid are the most common type of nutritional disturbance seen in the South. The symptoms of subclinical pellagra are quite similar to those of thiamine deficiency. Vague malaise, ready fatigue, anorexia, and constipation are common complaints. Glossitis and "indigestion" described as burning or sourness of the stomach occur rather early. The more severe psychic and somatic evidences of deficiency are apt to be precipitated by surgical procedures or by glucose maintenance. Delirium, stupor, various depressed or excited psychotic states[15], glossitis, stomatitis and diarrhea may be evidences of acute and probably almost total depletion of nicotinic acid in patients already in a poor nutritional state. The finding of a red atrophic tongue should always suggest nicotinic acid deficiency since this sign seems to be almost specific; delirium or stupor developing after operations may be the only evidence of nicotinic acid deficiency encephalopathy.

Methods are available for the chemical determination of thiamine, riboflavin and nicotinic acid in the blood and urine. Unfortunately these are of little diagnostic value since blood levels are apt to remain normal until the most severe grades of depletion are reached and the results of tests for urinary excretion are not apt to parallel clinical findings.

Inadequate intake of ascorbic acid is extremely common though clinical scurvy is quite rare. The symptoms of mild ascorbic acid deficiency cannot be differentiated from those of other vitamin deficiencies. Severe grades of depletion are characterized by definite evidences of increased capillary fragility, petechiae and ecchymoses occur from minimal trauma or spontaneously. Oozing from the mucous membranes may occur and there may be subperiosteal or intra-articular hemorrhages. Bleeding is not from any disturbance of coagulation but presumably from lack of coherence of the endothelial lining of the blood vessels. These effects of vitamin C deficiency are late and are slow to occur in the presence of an adequate supply of the other vitamins[7]. There are simple tests available for the determination of the concentration of vitamin C in the blood and for the rate of urinary excretion as well as for capillary fragility. Clini-

cal evidence of scorbutus may be present without chemical proof that vitamin C is absent from the blood and conversely, no clinical sign may suggest the deficiency when no ascorbic acid is present in the blood[8]. There is reason to believe that severe deficiency causes no definite symptoms or signs for a long while when other vitamins are present but that clinical scurvy may develop in the presence of polyavitaminosis long before the blood is entirely depleted of vitamin C. The presence of a positive test for capillary fragility requires that causes other than ascorbic acid deficiency be ruled out and also that the blood level and urinary excretion rate of the vitamin be determined.

Deficiency of vitamin K is to be suspected in all patients with a history of seriously deficient diet, in all with intestinal hypermotility and in every instance of biliary or hepatic disease.[16] The dietary sources of the vitamin are not abundant, absorption seems to depend on ample mixture with bile and the manufacture of prothrombin by the liver seems to depend on its functional integrity. Fortunately there are simple and rapid methods for the determination of the relative concentration of prothrombin in the blood which for clinical purposes can be interpreted in terms of vitamin K deficiency[17, 18].

Most of the recent literature on vitamin therapy records experimental observations of the effect of single vitamins on presumably specific manifestations of deficiency. The therapeutic aim in the management of surgical patients is altogether different, we attempt to prevent the development of any grade of nutritional disturbance or to cure any which may be present as rapidly as possible. These objectives are best accomplished by the use of a high vitamin diet and the administration of large doses of mixtures of synthetic vitamins supplemented when possible with small amounts of yeast or of liver extract. Patients fed through indwelling tubes can be given high vitamin liquid diet. Large daily doses of the water soluble vitamins should always be fractioned to avoid the loss by urinary excretion which occurs when single large doses are taken. Whenever there is reason to suspect that patients are in a state of nutritional inadequacy and certainly when there are any positive clinical findings of, or laboratory tests for avitaminosis, preoperative saturation should be attempted. Under ordinary circumstances this can be accomplished by oral administration of suitable preparations for four or five days. When nausea, vomiting, dysphagia or severe diarrhea are present intravenous or intramuscular injection must be used. If it is necessary to maintain patients on glucose solutions given by venoclysis the water soluble vitamins may be added to the solutions. No arbitrary dosage can be prescribed for preoperative

saturation but the amounts of thiamine, nicotinic acid and ascorbic acid used should be greatly in excess of those required for normal maintenance. We have had good results with 300 mg. of nicotinic acid, 15 mg. of thiamine and 3 mg. of riboflavin given in three doses during the day. Riboflavin may not be necessary in every case but it is my impression that it is useful. Ascorbic acid when used should be given in amounts of 600 to 1000 mg. a day divided into five equal doses. It is impossible to present statistical proof of the value of such a regimen but there is no doubt that the incidence of postoperative nausea, vomiting, gastrointestinal atony and delirium in depleted patients is greatly reduced. The use of 2-methyl-1, 4-naphtholquinone (synthetic vitamin K) is so general and the test for prothrombin so widely used as a preoperative precaution, that I mention them only to emphasize the fact that the B group vitamins are equally useful.

It is now evident that many of the more severe postoperative complications are of nutritional origin. I have already mentioned vomiting, gastrointestinal atony and delirium: it is notable that these occur more frequently after emergency operations than when patients have been properly prepared. These phenomena are very apt to be associated with a red, so-called "toxic" tongue and frequently with sore fissured lips and cracking of the mucosa and skin at the corners of the mouth. When for any reason patients are unable to take fluids or food by mouth and parenteral feeding with glucose is carried out for several days, similar changes in the lips and tongue may occur, delirium is apt to develop and not infrequently there is edema; this is usually regarded as "waterlogging" due to the sodium chloride content of the usual glucose solutions. These symptoms and signs are in no way different from those of endemic deficiency disease and respond rapidly to vitamin treatment. Glossitis and stomatitis, delirium and possibly vomiting seem to be due to nicotinic acid deficiency, edema frequently is discharged following the administration of thiamine, cheilosis is cured by riboflavin. It is not implied that all instances of postoperative complications of this type are of nutritional origin: infection with fever, partial obstructions of the bowel and drug intoxications may cause vomiting, distention or delirium; these very agents however are apt to result in severe avitaminosis with characteristic glossitis, stomatitis or cheilosis. Edema may result from hypoproteinemia or from excess of sodium chloride but in our experience these are less frequent than thiamine deficiency. Treatment of the severe deficiencies occurring after operations or during venoclysis must be vigorous and persistent, the requirements of some patients are tremendous. Because smaller amounts have sometimes been ineffective it is our custom to give from a gram to a gram and a half of nicotinic acid as

sodium nicotinate or nicotinamide, 30 mg. of thiamine and 5 or 10 mg. of riboflavin during the 24 hours for three days. These may be given in five equal doses at three or four hour intervals and either added to the infusion fluid or injected hypodermically. In those cases due to avitaminosis improvement is extremely rapid and it is usually possible to reduce the amounts of vitamins to ordinary treatment levels after the third day. Occasional patients with severe liver damage may require the larger amounts over a long period. It is my impression that small injections of the cruder liver extracts, 1 or 2 c.c. daily, are of distinct aid.

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THE RESULTS OF RADIUM TREATMENT OF CANCER
OF THE CERVIX

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MY FIRST impulse on receiving an invitation to address this distinguished body was to write down fully and frankly all the things that I do not know about cancer in general, and cancer of the cervix in particular. But then I was told that the time was limited. That settled it, for a far smarter man than I am, or even claim to be, would hardly get comfortably started, if he began honestly telling all that he didn't know about cancer, with only thirty minutes ahead of him.

Why, I don't even know what cancer is! I can't examine a patient and say whether my best efforts freely, conscientiously and intelligently given are going to be rewarded by a cure, or (what I prefer calling it) an arrest, or a complete failure. I can't do this even after a competent pathologist has diagnosed it and graded it.

The old pathologists used to say, "This is a nasty, virulent looking growth." Now they say, "This is Grade IV." But I don't know just what either of them means. What determines nastiness or high grades of malignancy in neoplasms? Are there different kinds of neoplasms, or do different people who happen to be the unwilling hosts to neoplasms react differently to the same kind of growth, thereby giving rise to all of these cellular differences in microscopic structures? I don't know. The streptococcus in an abrasion may result in a pipginny on one man's hand, and on another in a rapidly spreading cellulitis and septicemia that kills him. They don't react alike at all. The most learned investigators tell us, without reservation, that cancer does not come from without, it comes from within. Does it do the one, the other, or both? I don't know that either.

In my 164 cases of cancer of the cervix I haven't a single Jewish woman, although I have in my practice a fair proportion of Jewish patients. The incidence of Jewish women with cancer of the cervix is remarkably small. The Mayo Clinic reports "that in every 1000 non-Jewish patients seen by them, six have carcinoma of the cervix; in every 1000 Jewish patients only one has carcinoma of the cervix." Other clinics and statistical reports on large groups give approximately the same figures. But Jews have cancer of the breast, stomach and other organs just like other people. In the Fiji Islands there is a sect that practices circumcision as a religious rite, and the

wives of these people are as free from cancer of the cervix as are Jewish women, while the rest of the natives, who presumably are inducted into the church by the less realistic processes of sprinkling or deep baptism like us Christians, also share with us the same proneness to cervical cancer enjoyed by all true Aryans. This sort of thing looks like someone thinks that cancer may be an infection, and that all this is brought up to try to upset the authorities. It may not be convincing, but it is puzzling. After forty years of really intelligent thought I can't even say that cancer is a *cellular revolution* and not an *alien invasion,* nor can I affirm that the reverse is true.

Wouldn't it be a pleasing solution to the whole cancer problem if the degrees of badness, ugliness, nastiness or grading were all merely expressions of the patient's individual resistance, or lack of it, to some disease agent, just like in the pipginny and the septicemia patients who reacted so differently to the same streptococcus? I don't know but I am trying to do my own thinking. It is foolish for workaday people to be talking about doing research work. Leave it to the scientists and laboratory workers, and, if and when they discover the cause of cancer, a cure will soon be forthcoming. But while this is going on, we clinicians should bend every effort of mind and hand to see if we can better our results by keenness of observation and careful attention to detail in treatment.

I am talking about cancer of the cervix treated with radium. I am not decrying the use of x-ray as a substitute for radium, because I don't know enough about x-ray treatment alone to discuss it intelligently. I am using it in combination, or rather I am having it used, oftener each year, but I am carefully selecting the cases for the combined treatment and am still depending on radium alone in the greater proportion of our cases.

Prior to the use of radium we attained our best results from the use of the cautery. Our results following hysterectomy for cancer of the cervix were worse than bad. They all died, if not sooner, most certainly later. The hot iron relieved symptoms and prolonged life. They lived comfortably for a longer time than after operation. Of course the cautery destroyed the mass of cancer cells,—so would a knife or curette or scissors,—but the cautery did something more, just as it does in cancer of the skin and in advanced lip cases with alveolar invasion and where the bone too is invaded. Could it be that the heat kicked up a terrific reaction all around the growth and provoked the surrounding tissue cells to greater efforts at resistance? Bier demonstrated the usefulness of local hyperemia in infections. Why not something similar in cancer? Does anybody know what radium does to cancer? Does it kill the cancer cell, directly destroying it as the hot iron does? Or does it kick up some sort of a reac-

tion in the normal tissues that makes them more resistant to the cancer cell, or virus, or organism, or whatever it is that we call cancer? No one can say that this is true. Can anyone intelligently and knowingly deny it? I don't know, but I love to think about it.

I have read reams of literature on cancer of the cervix, and I must confess that not one of the hundreds of these authors tells me what I want to know. First of all, I want to know exactly how a case is managed from the time she comes to him until she gets well or dies, and how she dies, and what she dies of, if she does die. Then I want to know just what that particular writer thinks about the whole business, not what seventy-eight out of a hundred other writers think, but just what does he himself think. Believing that maybe there are a lot of simple-minded souls like myself who would like to know the things I'd like to know from other people's work, I am at the very real risk of seeming tedious and elementary, going to tell you how I handle a case from start to finish.

First a complete history is taken as in any other case. Then a careful physical examination is made including a pelvic and a biopsy if the diagnosis is doubtful. If it is manifestly cancer the biopsy is done at the first application of radium. All these data are then set down upon a cancer card so that the same essential facts may be stated clearly about each patient in its own particular column, and by adding to these from time to time the entire picture of each case can be condensed upon one small card.

Our plan is to give from 6000 to 8000 milligram hours of radium to each patient. We divide this into three monthly applications, thus allowing about thirty days for the last reaction to subside before giving the others. We nearly always give a general anesthetic with the first application and usually with the others. We use the cautery when there is a large bleeding mass. By removing this with the hot iron we lessen the work of the radium and place it higher up toward the high tide mark.

When the lesion is small enough to remove radically along with the lower cervix, we do so and apply radium above the high tide mark.

I believe that good results can be obtained with a comparatively small amount of radium. We use only 100 milligrams. It is encased in glass, platinum and brass just as it comes from the dealer. Our initial dose usually is given as follows: A 25 mg. container is encased in rubber and passed into the fundus. Fifty milligrams similarly covered is placed in the cervix. It is held in by wrapping a coarse thread around the outside of the rubber. This makes it as difficult to slip out as if it were a screw. Then 5 needles of 5 mg.

each are plunged into the diseased cervix at such points as the covering of the invaded area seems to require. We usually leave these in for 36 hours, thus giving 3600 mg. hrs. In a month we give from 2000 to 2400 mg. hrs, and the same at the third treatment.

What do we look and hope and pray for after the first application of radium? A cessation of bleeding and regression of the growth, a shrinkage, a subsidence, an attempt at a return to normal shape, size, and appearance. Now this should be progressive after each treatment. And in from two to three months after the last treatment, the condition should approach a normal, small, atrophic uterus and cervix such as might be found in any elderly woman. These cases should be watched by someone,—every three months for the first year, every six months for the next two or three years, and once a year as long as they live. We take the body weight, hemoglobin, the red cell count, and the appearance and feel of the pelvis as criteria of improvement, a yardstick so to speak, to measure by.

But what does a bad case do and why does it do it? Some cases continue to bleed in spite of the radium. I have sometimes found heavy x-ray treatment here most helpful, at other times not. Instead of regression we often get sloughing. There is a funnel-shaped crater that grows and grows. It has a diphtheritic membrane upon it; it advances or creeps inexorably but slowly; it gives rise to fistulas, urinary and fecal. The end comes slowly and with great pain and misery and pitifulness.

I'd love to be the man who knows how to turn this kind of case into the first kind. Both cases might have started out looking just alike as to class and grade. If only we had the sense to bend or twist them back into that other channel, and to make them behave in an orderly manner. I'd rather be that man—

There are a few concomitant conditions that may affect the outcome of a case most seriously,—syphilis and diabetes,—and I'd rather have the former to deal with than the latter.

Among the complications arising during treatment are atresia of the cervix, cystitis, pyelitis, stricture of the ureter and rectum, and fistulas. Atresia can be easily dealt with by dilatation of the cervix and irrigation of the uterine cavity with saline solution. Cystitis, pyelitis, and ureteral strictures are treated in the manner laid down by urologists, only with these cases the trouble is more stubborn with so many structural changes and lowered tissue and general resistance. When we take cases as they come, good, bad, and indifferent, advanced and more than advanced, some of them are bound to slough into the bladder or rectum, whether they are treated or not.

There was a young Englishman named Thomas Francis Todd killed in France last December. It is a pity; a lot of knowledge died with this young man. He wrote most clearly, completely, and sensibly about the resulting stricture of the rectum after radium and x-ray, and the connective tissue changes that often freeze and distort the whole true pelvis.

I have a big strong woman in her twelfth year with a moderate sized vesicovaginal fistula. She is also a high grade diabetic. She feels perfectly well and says she does not want to risk an operation for its repair as she has quite adjusted herself to it. Maybe she is right. I have another woman who has a fistula of the rectum into the uterus. The rectum is strictured and carries only a part of the fecal matter off. She is in her fourth year and I am trying gradually to dilate her rectum. The uterine sphincter seems to be showing a remarkable adaptability for its new function, and the patient looks well and seems quite happy as she is. After all a fistula is far preferable to a cancer. One may be able to adjust one's self to a fistula but never to a cancer.

The patients represented in this group were all private cases and only a few were treated out of our own hospital. My earlier work was done in collaboration with the late Dr. Samuel Budd, and this continued until his death, July 27, 1938. Dr. Budd believed that dark women, brunettes, and especially those approaching the masculine type, were more resistant to cancer than their fairer more feminine sisters. Although we have but a few figures to base our opinions on, we are still studying this aspect of our cases more thoroughly, hoping that more data may mean more light even if just a little more. I remember a stout old lady with coarse dark hair and with a good mustache. She came to be examined regularly every six months after her first treatment. She missed one visit and came at the end of her eighth year. She had a bad break that nothing would stop. She died quickly. She lived long enough to have one five year cure, and to have gotten well past the half post of a second five year cure. I remember two blond women, one with adenocarcinoma of a left-in cervical stump, the other a typical epithelioma of the intact cervix. Both healed beautifully and neither could be induced to have regular examinations. Both had recurrences with splendid results on additional irradiations. Improvement lasted from six months to a year. In the end both died miserably of extension into the pelvic viscera. I believe that these two women could have been kept approximately normal over a period of many years had they been closely watched, examined and treated.

What is going on in the tissues of these women who seem to have been completely cured, who are completely healed? Have we left

something, a nidus or island, within the healed tissues, a spark that something may blow into a consuming fire again. How can we put out all the sparks and know that they are out? What is it that blows them into flame? How can we stop it?

If there were just some way to pick these people out beforehand. If we only knew at what interval to give them some protective treatment, what and how much, we would put a few more of our cases on the black side of our ledger column. But we do not know, and the only alternative is to watch them all, insisting on regular, thorough examinations of every one of them, so as to detect the break as early as possible and to treat it vigorously as soon as it is recognized.

I am convinced that there is something in Budd's observation about these brunettes. Maybe something can be done by the endocrine people, inducting the feminine type into the masculine type or in some way approaching such a transition. I mean something deep and fundamental that would reach into the warp and woof of her and shake the very chromosomes of her fibres. The beauty parlor people are but superficial at best.

I have a feeling that concomitant infection plays a much more important role in cancer of the cervix than most of us realize. Meticulous care in keeping the growth clean may well prove one of the determining factors in the recovery of some cases. Chemotherapy will almost certainly be of the greatest help by turning infected cases into simple cancer cases when we know just a little more about the choice of the drug to be used.

Now about the curability or arrestability of these cases. Our records show that we have treated or have under treatment 164 patients. Eliminating those of whom we have lost trace before their first 5 year period had been completed, and those who died of other diseases before this period was completed, we have a total of 121 cases left. Forty-four of these, or 36 per cent, are recorded as arrests or cures. Our previous reports showed a percentage of recoveries as follows: In 1927, 29.5 per cent; in 1932, 31.5 per cent; in 1934, 32 per cent; and now, in 1941, 36 per cent. So it can be seen that our figures at least show a steady if small improvement.

The astounding thing to me is that the results of widely separated operators, treating large and small groups of these cases, should be so nearly alike. There is something fixed about it, as fixed as a roulette wheel where the house takes 67 and the player takes 33. Suppose 100 cases would all present themselves at once, just in the state that they would be in, had they come normally through the years, good, bad, and indifferent. Sixty-seven of them are going to

die and thirty-three of them are going to get well provided they get the best possible care. Even by the most careful examinations, could you sort them out beforehand and put them in their places? I know that I could not. I'd condemn some awful looking ones who would make good recoveries; and I'd be bitterly disappointed and distressed by some early cases dying in the first and second years.

.What is an early case? Three women have come to me by ambulance for devastating hemorrhage. They arrived in a few hours after their very first symptom. Were they early cases? How could they have been gotten earlier? So many cases, single cases, and groups of cases, bring you up against a solid brick wall. If only we could pick out some of the bricks and make a hole to peep through and see what's on the other side! If we never stop trying, never get discouraged, and keep doggedly at it, we may get just an occasional glimpse, and soon a few more, even one, two, or three more each year may reward the earnestness of our efforts.

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The
Southern Surgeon

DR. ANSON JONES,
Last Texas President

Southern doctors can point with pride to the enviable records and often great distinctions of their confreres in pioneer communities. The professional training of these men seemed to make them peculiarly adapted to the physical hardships and intellectual demands of the raw frontier settlements. Nowhere more than in Texas were the early doctors more zealous in their contributions to the establishment of order and well-being. And in the growth of Texas no one achieved such eminence and fame as Dr. Anson Jones who led the early Republic into the safety of Statehood. When on February 19, 1846, he furled the lone star flag of Texas and said, "The final act in the great drama is now performed. The Republic of Texas is no more," he simultaneously completed one of the most spectacular careers of any member of our profession.

Anson Jones began his life January 20, 1798, at Great Barrington, Massachusetts, and ended it like many contemporary Texas notables by suicide in Houston, January 7, 1858. His elementary education was gained at Lenox Academy near his birthplace and completed in 1815. Two years later while his father and older sisters pressed him to study medicine he says, "I shrunk from the idea of making the effort to obtain a profession, and feared I should not succeed in it afterwards—which last thought the most troubled me." Nevertheless in 1817 he began to read medicine with Dr. Daniel Sheldon of Litchfield, Massachusetts, and was assigned such uninspiring reading as Boerhaave's *Aphorismi* and *Institutiones*. The next year he studied in the office of Dr. Amos G. Hull of Utica, N. Y., and in 1820 was licensed to practice by the Oneida Medical Society.

Dr. Jones attempted to establish a practice in Bainbridge, in Norwich (where his stock of drugs was seized) and in Philadelphia (where he was arrested for debt). Such unglamorous failures caused him to leave New England with few regrets and sail to Venezuela. During the next years in the tropics he saved enough money to return to Philadelphia, to complete his medical course at Jefferson Medical College, and to receive the M. D. degree from that institution in March 1827. Again he tried to practice in Philadelphia but once more he was overtaken by debts.

Dr. Jones moved to New Orleans to attempt running a mercantile business. This also failed so: "in the spring of 1833 I opened an office on Canal Street for the practice of my profession. The summer proved very sickly, and I was succeeding as well in my business as I could reasonably expect . . . " Then he became ill while the health of New Orleanians improved so much meanwhile that his practice dwindled. Moreover he made an early observation on New Orleans which scarcely needs documentary corroboration: —"I found the pernicious habit of gambling, to which I always had an inclination, was growing upon me there . . . the constant temptation thrown in my way I found was slowly overcoming my resolutions. . . . Whilst in this place also, partly from having frequently little else to do . . . I found myself learning to imitate the fashionable practice of taking a 'julep' much oftener than was at all necessary . . . I therefore felt anxious to get away from the place and its associations."

A ship captain interested Dr. Jones in Texas and told him "there was a good opening for a physician at Brazoria." He promptly moved to Brazoria in the new colony and gradually developed a successful practice while at the same time he became aware "that somehow or other the destiny of Texas was interwoven with my own, that they were indissoluble, and that the one depended materially upon the other." The sixteen succeeding years fully justified this early impression gained by the young doctor.

When Dr. Jones moved to Brazoria his assets included 17 dollars in cash, 50 dollars worth of medicines, and old debts of $2000. However, "having concluded to give Texas a trial, I immediately commenced the practice of my profession at Brazoria and soon took the lead of all competitors of that county." By the end of 1834 he was handling a practice worth $5000 a year and was well on the way to "become worth an independent fortune and as wealthy as any man in Texas." But the premonitory signs of revolution were appearing and Dr. Jones soon became so engrossed in the public affairs of the colony that he was compelled to discontinue practice permanently.
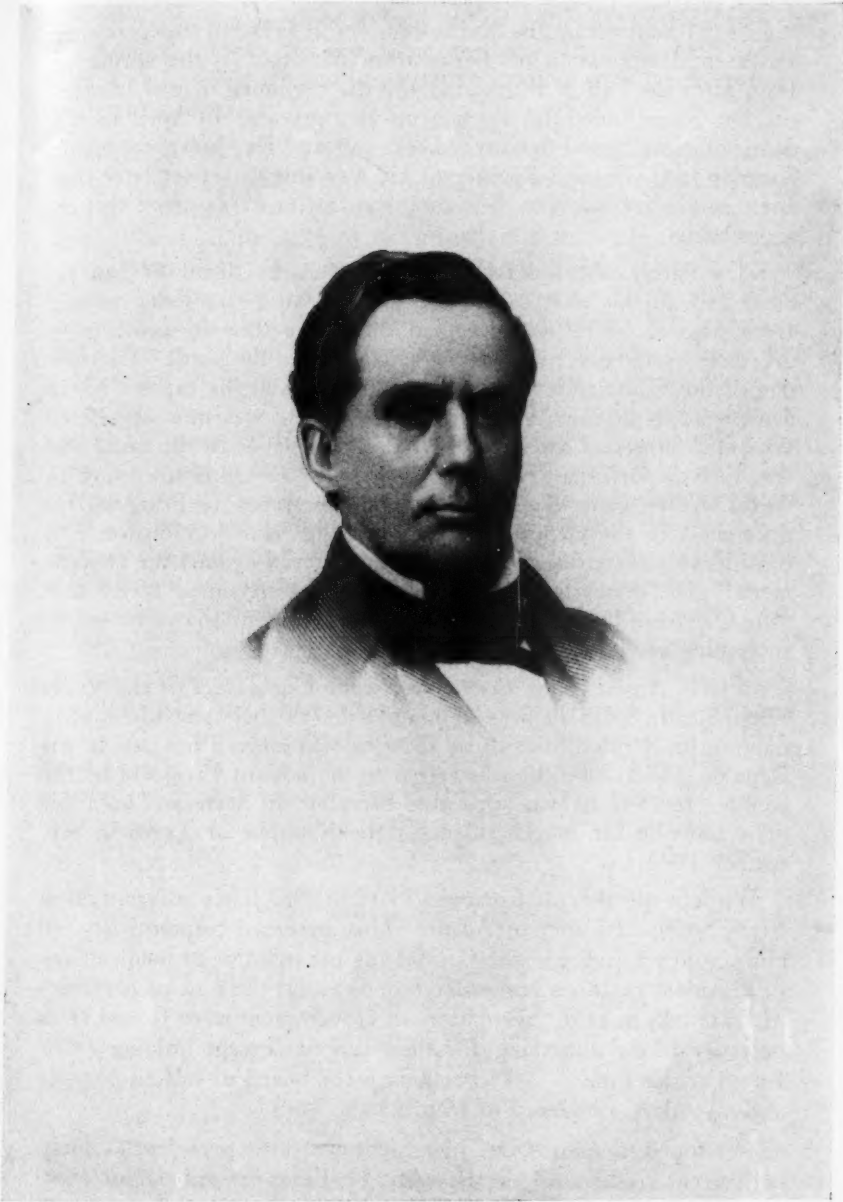
Anson Jones, M. D., 1798-1858

In 1835 he drew up the first resolutions in favor of independence and a total separation of Texas from Mexico. In the spring of 1836 after the Fall of the Alamo and the beginning of war in earnest, Dr. Jones joined the Texas army as a private. In April an epidemic of measles and dysentery broke out and Dr. Jones was made Surgeon to the Second Regiment. He was able to report later that not a single life was lost in the regiment after he assumed this responsibility.

Immediately afterward Dr. Jones was in the Battle of San Jacinto and did his part to aid the winning of Texas independence from Mexico. He "was occupied the entire time in assisting to dress the wounds received on the field." After the battle "I accompanied the Commander-in-Chief (Houston) and the captive Mexican president (Santa Anna) to Galveston. I was now appointed Assistant Surgeon General and Medical Purveyor to the army and sent to New Orleans to procure supplies. . . . " Upon his return to Texas the Surgeon General called on Dr. Jones to bring all his documents to the Capitol because "the Old Man" (Houston) "is considerably exercised about complaints entered against the Department." Dr. Jones ignored the order and commented wryly that "the Old Man had better get sober and attend to affairs he knows something about."

In 1837 Anson Jones was elected to the Legislature of the Texas Republic. In 1838 he was appointed Texas' Minister-Plenipotentiary to the United States. In 1838 he was elected Senator in the Republic and in 1840 he was raised to the post of President of the Senate. In 1841 he was appointed Secretary of State and held this office until he became President of the Republic of Texas in September 1844.

While a member of Congress in 1840 Dr. Jones married Mrs. Mary Smith McCrory of Austin. This increased responsibility led him to open an office in the Capitol for the practice of medicine until President Houston persuaded him to accept the post of Secretary of State. As he said, "the officers of Government were forced from necessity to do something for their support except holding office. I paid at this time . . . $75 per month for board of self and family and my salary amounted to from $50 to $60."

For fourteen months Dr. Jones performed the presidential duties with great credit until the Republic of Texas passed out of existence and was replaced by the State of Texas. His diplomatic and domestic achievements were very striking in a world that was in great turmoil. He kept Texas at peace, parried foreign efforts to control the Republic, balanced the budget and preserved for the

new State the unique right to keep all her public lands when joining the Union. It was this last achievement which gave the State of Texas and not the Federal government control over large areas of oil-bearing lands.

When Texas became a State Dr. Jones felt that he could at last realize the retirement he had so long sought and the abandonment of public life. He moved his family to his farm "Barrington" and began the quiet pursuits of the rural gentleman. In 1849 he received a crushing injury of one forearm after a fall from a vicious horse, and this further confirmed his decision to live quietly in his declining years. Still his friends would not respect his peace and in 1857 they induced him to run for the United States Senate. He was roundly defeated and bitterly disappointed. Shortly afterward he talked to a friend in the Old Capitol Hotel in Houston and remarked, "Here in this house twenty years ago I commenced my political career in Texas as a member of Congress and here I would like to close it." An hour later he shot himself as had many others in that lusty early period. A General of the Army, the first Chief Justice, the author of the Texas Declaration of Independence and a prominent candidate for the Presidency of the Republic had all seen fit to take their own lives after experiencing reverses.

Dr. Anson Jones, the southern surgeon, Surgeon-General, patriot, diplomat and Texas President, excites our interest as one of our confreres who was eminently successful in many fields. As an early "political doctor" he set a standard of excellence which has seldom been equaled.

—WALTER G. STUCK, M. D.

The Editors of THE SOUTHERN SURGEON desire to record the military activities of the Fellows of The Southeastern Surgical Congress and of the Texas Surgical Society. They request each man who is called to active service to notify them so that the fact may be recorded in these pages and the Journal may follow him.

Lieut. F. P. Holder, Jr., of Eastman, Ga., has been on duty with the Fifth Medical Battalion at Fort Custer, Mich., since Dec. 5, 1940.

Capt. Kenneth R. Bell, of Sanford, Fla., has been serving as Assistant Chief of Surgical Service at Station Hospital, Camp Claiborne, La., since February 1.

Capt. E. W. Cullipher, of Miami, reported for duty with the Army on February 28 and, after three changes of station, is now at Billings General Hospital, Fort Benjamin Harrison, Ind.

Capt. Samuel R. Terhune, of Birmingham, has been on duty at Station Hospital, Camp Polk, La., since April 1.

Lieut. J. G. McDaniel, of Atlanta, has been stationed at the U. S. Naval Hospital in Pensacola, Fla., since May 19.

Lieut. Hub E. Isaacks, of Fort Worth, is on active duty at the Station Hospital, U. S. Naval Air Station, Corpus Christi, Texas.

THE INTERNATIONAL ASSEMBLY OF SURGEONS
MEXICO CITY, AUGUST 10 TO 14.

The Mexican Ambassador at Washington in July requested the Secretary of State to transmit to interested organizations and individuals in the United States an invitation on behalf of his Government to participate in the International Assembly of Surgeons which will be held at Mexico City from August 10 to August 14, 1941.

This invitation was received at the headquarters of The Southeastern Surgical Congress too late for a full list of delegates to be published in this number. Those who have already been able to arrange to accept this invitation of the Mexican Government are Dr. Herbert Acuff and Dr. B. T. Beasley.

BOOK REVIEWS

INFANTILE PARALYSIS. By PHILIP LEWIN, M.D., F.A.C.S., Associate Professor of Bone and Joint Surgery, Northwestern University Medical School. 372 pages, with 165 illustrations. Price, $6. Philadelphia: W. B. Saunders Company, 1941.

Some fifty cases of acute anterior poliomyelitis having broken out in the home town of the reviewer during the past month, this new book has proved of peculiarly vital interest to him.

Dr. Lewin has not set out to produce a work of reference for specialists in virus research, epidemiology, immunology or orthopedic surgery, but these subjects are covered adequately for the average physician. His aim has been to enable doctors, especially the general practitioner, to recognize the disease in the preparalytic stage and to start the proper course of treatment. His thesis is "No major deformity should be allowed to occur."

The reviewer feels sure that 170,000 doctors in the United States could learn from this book a great deal they don't know now about infantile paralysis, a disease which, though not very common, is responsible for so much crippling of young people, crippling which can be in large part prevented.

FRACTURES AND OTHER BONE AND JOINT INJURIES. By R. WATSON-JONES, M.Ch.Orth., F.R.C.S., Consultant in Orthopedic Surgery of the Royal Air Force; Member of War Wounds Committee Medical Research Council; Lecturer in Orthopedic Pathology and Clinical Orthopedic Surgery, University of Liverpool. Second edition. 724 pages, with 1040 illustrations. Price, $13.50. Baltimore: The Williams & Wilkins Company, 1941.

Since the 1870's exceptionally good orthopedic surgery has flourished in Liverpool where the late Sir Robert Jones popularized the teachings of his uncle Hugh Owen Thomas at the well-known Rodney Street address. The present-day orthopedic surgeons in Liverpool have ably carried on this great tradition despite repeated bombings and Watson-Jones' book is a good demonstration of masterful work performed under trying physical hardship.

Even though American orthopedic surgeons now contribute most to the specialty, their English contemporaries have continued to display unusual ingenuity and skill. Watson-Jones has carefully selected one of the most complete expositions we have on the subject.

Instead of the usual fracture text which follows the time-worn pattern of describing treatment of all the common fractures, this book includes many additional inseparable subjects. The first section on the phenomena of fracture repair contains a vast amount of information since Watson-Jones is one of the great authorities on calcium metabolism. Also the chapters on war wounds and pathologic fractures are extraordinarily complete. In addition to a thorough explanation of treatment of all types of fractures there are chapters on back injuries, knee derangements, nerve injuries, vascular accidents and such other related traumas which the surgeon may encounter. Also discussions of

rehabilitation of the injured and repair of malunited fractures are unusual in this type of book.

All the descriptions are concise and an unusually large number of illustrations (1040) makes the points easily understandable. A critic might point out that the author is dogmatic and very positive in his statements. Yet this is not undesirable when the author is a great authority (this book is based on an experience of over 47,000 bone and joint injuries) and has added the opinions of the best surgeons throughtout the world.

This is a reference work wherein the surgeon can readily find the most suitable method of treatment of nearly every type of bone and joint injury. It will be invaluable to him when he seeks expert advice on controversial matters relating to trauma.   —W. G. S.